PLANT PATHOLOGY

A RECORD OF CURRENT WORK ON PLANT DISEASES AND PESTS

Published quarterly for the
Ministry of Agriculture, Fisheries and Food
Plant Pathology Laboratory
Harpenden, Herts

VOLUME 6, No. 4
December 1957

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WIREWORM AND SLUG DAMAGE TO WARE POTATOES, 1954-56

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Each autumn since 1947, the Potato Marketing Board (Potato Division of the Ministry of Food before 1955) has carried out a survey of maincrop potato growers in order to obtain good, pre-harvest estimates of ware production. Fields surveyed were, in general, on the same farms each year, and the original farm sample was selected to be as representative as possible of potato crops in each region of Great Britain having regard to variety, soil type, and potato acreage. Growers of less than one acre of potatoes have not been fully represented in the annual samples since 1955 because the Marketing Board could only obtain information from registered producers and registration was not required of those who set less than one acre.

Since 1952, advantage has been taken of the crop check-weighing scheme to obtain information on the incidence of Common Scab, and in the report on this work in 1952 and 1953 (Large and Honey, 1955) the standard yield sampling method is described. Latterly, the sampling rate has been varied such that 4 samples are taken on fields between 3 and 10 acres in size, whereas up to 8 are taken in fields of 30 acres or more. In 1953, information was wanted on insect pest damage to the ware crop, and in 1954 and the two following years the crop check inspectors were asked to obtain supplementary information on wireworm and slug damage in each of the fields on the sample lists. In practice, nearly 3,000 ware crops, representing about 4 per cent of the national potato acreage, were inspected each season, and the sample tubers were graded as " sound " (those standing on a 1½-inch riddle which the trade would accept as ware), " wireworm damaged " (those with sufficient wireworm holing to render them unsaleable on the ware market), and " slug damaged ". In the rather rare cases where both types of damage occurred on the same tubers, grading was according to the pest responsible for the greater part of the damage.

ACCURACY OF THE DAMAGE ESTIMATES

The accuracy of the crop check yield sampling method is known to be satisfactory. A standard error of rather less than $\pm$ 0·2 tons per acre on an average yield of about 8·5 tons per acre was found by Dyke and Avis (1953) when a similar method was used on a sample of 1,000 fields in England and Wales. There remain, however, three sources of error in the detection of pest damage within the yield samples : first, tubers lifted from moist or wet ground are often covered with a thin layer of adherent soil which can obscure traces of insect damage ; second, the crop check survey work necessarily has to be carried out ten to thirty days before the crops are lifted and cannot therefore give a true picture of wireworm damage at the time of lifting ; and third, if, as Miles and Cohen (1937) have suggested, wireworm injury continues in clamped tubers, a further source of error is introduced in the assessment of national losses from crop check survey results.

The first of these sampling errors was investigated in some detail. The Marketing Board fieldmen had been asked to make a subjective assessment of the pest-damaged ware they found in their samples. If, in their individual opinions, the tubers were not damaged sufficiently to affect the sale of the crop as ware then those tubers were to be classified as sound. From an entomological point of view, however, even superficially-holed tubers cannot be regarded as sound, so it was necessary, in the first place, to relate the subjective assessments to a more precise objective standard. In addition, it was obviously necessary to find out the extent to which moist adherent soil prevented an accurate assessment of damage.

The co-operation of the advisory entomologists in the National Agricultural Advisory Service was sought and it was arranged that an entomologist in each of several areas should accompany Marketing Board teams on visits to fields expected to show wireworm damage. In the event, reliable data from only three fields were obtained, and these suggested that the entomologists were grading the tubers more severely than the fieldmen, the percentages of tubers damaged being 26 and 18 per cent respectively. These results did not agree with those obtained in 1953 when complete crop check samples from fifteen fields in Yorkshire, Nottingham, Lincolnshire, Leicestershire, Essex, Berkshire, Oxford, and Gloucestershire, were submitted to the Plant Pathology Laboratory for examination after assessment in the field by Marketing Board staff. The entomologist at Harpenden rejected an average of $3{\cdot}6 \pm 0{\cdot}43$ lb of tubers per sample as pest damaged, compared with $5{\cdot}0 \pm 1{\cdot}26$ lb per sample rejected by the fieldmen. In the absence of other information it seems that there is no very substantial difference between the two types of estimate of field damage.

In 1955 and 1956, the advisory entomologists examined samples of tubers in the field at nine sites and on two sampling dates and then took the same tubers back to the laboratory and re-examined them after careful washing. Whereas the field examinations suggested that an average of 30 per cent of the tubers were damaged by wireworm, washing showed that 40 per cent were, in fact, damaged, a difference which is significant at the 5 per cent point. The results from eight sites visited in 1956 were further analysed by classifying the sites into those with slight damage (up to 0·8 tons of ware per acre damaged by wireworm), with moderate damage (0·8 to 1·99 tons per acre), and with severe damage (2 tons or more per acre). The slight and moderate categories showed a significant difference between the amounts of damage noted on the washed and unwashed tubers, whereas the severe category showed a similar, though non-significant, difference. On the limited data at present available, therefore, it seems that visual examination of freshly-lifted tubers in the field enables about three-quarters of the wireworm damage to be detected. Slug damage, which is generally grosser than wireworm holing, is quite easy to detect without washing.

In order to assess the increase in wireworm damage between the time of crop check and lifting, fourteen fields in different parts of the country were revisited by the fieldmen at lifting in 1955, and the level of damage they found then was compared with the level found at their first visit (on both occasions the tubers were examined unwashed in the field, and a direct comparison can therefore be made between the two visits). An average of 12 per cent of the tubers showed damage by wireworm at the time of the survey, and by lifting time 23 per cent of the tubers were holed. In 1956, comparable figures were obtained from one site only, 26 per cent holing being found at the first visit compared with 46 per cent sixteen days later when the crop was lifted. From the two years' results

it seems that damage is almost doubled between crop checking in September and lifting in October.

Increases in damage in storage were investigated in 1955 and 1956 by Marketing Board staff in collaboration with advisory entomologists. Most of the stores visited were clamps, but by 1956 indoor storage had become popular and of the ten stores examined in that year four were in barns. No information was available on clamp or store sampling and an arbitrary scheme was introduced in which a minimum of 100 tubers were removed from each of three sampling stations, one at random in each third of the clamp or heap. These sampling stations were visited at clamp making and subsequently at stated intervals through to clamp breaking. In 1955, most of the samples were examined straight from the clamp. From five crops examined at lifting and again when the tubers had been in the clamp for about two months, damage estimates of 43 and 51 per cent respectively were obtained. Results from six crops examined after being clamped for about six weeks and again after about three months suggested that damage increased from 56 to 60 per cent, but data from four crops examined at clamp construction and again at clamp breaking indicated no increase in damage. It was felt that this apparent overall slight increase in damage during the first few months in the clamp may have been due to adherent soil, which had dried and fallen from the clamped tubers, making wireworm holes easier to see.

To overcome this source of error in 1956, tubers were first examined unwashed straight from the clamp, and then taken back to the laboratory, washed and looked at again. The first visit was in October or, in one case, in November, and the second was either in December or in January. No evidence for an increase of wireworm damage during storage was found. In fact, the average percentage of visible damage on washed tubers on the first date was 43 per cent, and on the second date 39 per cent. A similar comparison was made at four sites where counts were made in December or January and again in March, and no increase in damage was found. Taking into consideration the available results from both years, there is no evidence of appreciable increase in damage within the clamp or store ; the evidence so far available suggests that crop check estimates should be doubled if a reliable estimate of actual economic damage at lifting or clamp breaking is required, this being due almost entirely to late-autumn wireworm feeding in the two or three weeks before lifting.

NATIONAL LOSSES DUE TO WIREWORM AND SLUG DAMAGE

In the last few years there has been only a very small commercial tolerance of damaged ware. In years when the potato crop is good, no damage at all is tolerated, and any ware crop affected by wireworm or slug has to be sold at a considerably reduced price as stock feed or for industrial purposes. Account has been taken of this situation in analysing the crop check survey results in that pest damage has been divided into three arbitrary groups, slight, moderate, and severe damage, defined earlier, in respect of the quantity of tubers standing on a 1½-inch riddle. With crops classified as slightly damaged it is generally worth while for the grower to riddle out the affected tubers so that the bulk of the crop can be sold as ware ; moderately damaged crops might be riddled through in a season when ware is in short supply, but it is very unlikely that crops in which pest damage amounts to 2 tons or more per acre are ever offered as ware.

Total ware production in Great Britain for the three years in which information on pest damage has been obtained is shown in Table 1. Although the tonnage of ware produced has varied considerably over the three years, the acreage planted has remained much more constant.

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TABLE 1

TOTAL WARE PRODUCTION, GREAT BRITAIN, 1954–1956
('000 tons)

N.A.A.S. Region	1954	1955	1956
Eastern	1,222·8	1,119·0	1,338·7
South Eastern	248·6	232·8	315·7
South Western	169·3	175·6	250·3
West Midland	395·9	318·1	452·8
East Midland	831·6	628·3	818·4
Yorks and Lancs	575·4	500·7	568·9
Northern	360·1	304·4	342·3
Wales	119·2	97·7	136·0
England and Wales	3,922·9	3,376·6	4,223·1
Scotland	1,022·3	898·5	1,008·5
Great Britain	4,945·2	4,275·1	5,231·6

Table 2 shows the gross losses due to wireworm attack on the ware crop in the three years for which information is available. Individual field estimates have been used in the calculation of these losses, which refer to the total quantity of ware from all fields on which any level of wireworm damage was detected at crop check. In a sense they define the upper limit of wireworm damage because they include both sound and damaged tubers from the fields in question. As has been suggested before, in practice the grower will often riddle out the damaged tubers from slightly attacked crops, and sometimes he will do the same with a crop showing moderate damage at lifting.

TABLE 2

ESTIMATED TOTAL TONNAGE OF WARE DAMAGED BY WIREWORM, 1954–1956
('000 tons)

	Slightly Damaged			Moderately Damaged			Severely Damaged		
N.A.A.S. Region	1954	1955	1956	1954	1955	1956	1954	1955	1956
Eastern	96·5	13·9	28·4	0·0	0·0	0·0	5·6	1·4	2·5
South Eastern ..	33·0	10·4	16·9	10·9	0·8	3·3	2·3	0·0	0·0
South Western ..	14·6	4·4	12·9	7·8	10·1	6·8	11·9	1·8	8·8
West Midland ..	25·8	11·4	29·1	17·8	0·0	8·3	1·1	1·3	0·0
East Midland ..	41·0	46·1	49·9	9·1	0·3	2·8	2·8	2·1	1·3
Yorks and Lancs ..	22·3	1·5	0·6	1·9	0·8	0·0	8·2	4·4	1·8
Northern	61·9	25·7	16·6	1·7	4·9	7·4	0·0	1·9	0·0
Wales	6·5	4·5	9·3	3·8	0·3	8·6	0·6	0·0	0·0
England and Wales ..	301·6	117·9	163·7	53·0	17·2	37·2	32·5	12·9	14·4
Scotland	19·1	6·1	5·3	1·8	0·0	0·0	0·0	0·0	0·0
Great Britain ..	320·7	124·0	169·0	54·8	17·2	37·2	32·5	12·9	14·4

If the three-year average is a fair estimate of the situation prevailing in a normal year, it seems that gross wireworm damage accounts for about 250,000 tons of ware on a normal production of 3·84 million tons in England and Wales—or about 6·5 per cent of the crop. There is, however, considerable variation in the amount of wireworm damage recorded from year to year, losses in 1954 being more than twice as heavy as in 1955, and, in spite of increased insecticide usage (Table 7), damage in 1956 being greater (5·1 per cent of total production in England and Wales) than in 1955 (4·4 per cent of production). Scottish growers appear to suffer less from wireworm trouble than those in England and Wales and, on the evidence available, it seems that severe damage is uncommon north of the Yorks and Lancs Region.

Wireworm and Slug Damage to Potatoes

Losses due to slug damage are shown in Table 3. They follow much the same pattern as wireworm losses. The exceptionally wet autumn of 1954 may have influenced the heavy infestations which damaged nearly half a million tons of ware in that year; and the hot, dry, summer of 1955 may well have had an adverse influence on slugs as well as on potato yields, leading to a considerable decrease in gross losses.

Table 3

Estimated Total Tonnage of Ware Damaged by Slugs, 1954–1956
('000 tons)

N.A.A.S. Region	Slightly Damaged			Moderately Damaged			Severely Damaged		
	1954	1955	1956	1954	1955	1956	1954	1955	1956
Eastern	147·4	126·2	44·5	2·4	0·0	1·9	0·0	0·0	0·0
South Eastern ..	18·4	12·0	21·8	2·2	0·0	1·2	0·0	0·0	0·0
South Western ..	15·2	4·8	3·0	0·0	0·0	0·0	0·0	0·0	0·0
West Midland ..	16·5	2·9	13·6	1·7	0·0	0·0	0·0	0·0	0·0
East Midland ..	75·9	50·6	62·1	1·8	0·0	0·0	0·0	0·0	0·0
Yorks and Lancs ..	9·5	30·1	20·3	4·0	4·5	2·0	0·0	0·0	0·0
Northern	23·6	10·4	19·2	1·4	0·0	0·0	0·0	0·0	0·0
Wales	7·6	3·9	7·4	0·9	0·0	0·0	0·0	0·0	0·0
England and Wales ..	314·1	240·9	191·9	14·4	4·5	5·1	0·0	0·0	0·0
Scotland	72·0	0·0	71·1	3·4	0·0	3·1	0·0	0·0	2·6
Great Britain ..	386·1	240·9	263·0	17·8	4·5	8·2	0·0	0·0	2·6

Slug damage of a severity sufficient to render a crop unsaleable on the ware market is less common than severe wireworm damage, though slugs seem to be more important than wireworms in Scotland. Gross slug damage in England and Wales accounts for about 6·7 per cent of the crop in a normal year, though there is obviously a large error in attaching this average value to any one year.

Table 4

Corrected Net Estimates of Wireworm and Slug Damage, 1954–1956
('000 tons)

Losses	England and Wales			Scotland			Great Britain		
	1954	1955	1956	1954	1955	1956	1954	1955	1956
Due to wireworm	90·5	37·3	53·7	0·9	0·4	0·3	91·4	37·7	54·0
Due to slug	11·6	5·5	5·0	2·6	0·0	1·9	14·2	5·5	6·9
Allowance for damage by both pests together ..	6·3	2·7	4·4	0·0	0·0	0·1	6·3	2·7	4·5
Final loss due to both pests	95·8	40·1	54·3	3·5	0·4	2·1	99·3	40·5	56·4

The damage estimates given in Tables 2 and 3 are gross, or maximal, estimates of loss. By taking the actual quantities of damaged tubers per sample for individual fields, it has been possible to prepare minimal estimates of loss—that is, estimates of the actual quantity of tubers with pest damage as distinct from the bulking of sound with damaged tubers in respect of each field on which damage occurred. It has already been suggested that the crop check estimates of damage are minimal in the sense that further damage usually occurs before lifting takes place, and the limited evidence available suggests that by doubling the crop check figures a fair estimate of actual commercial damage can be obtained. Table 4 has therefore been prepared in the form of corrected net estimates of the actual commercial losses due to the two pests under consideration, on the assumption that crops with less than 2 tons per acre of damaged

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tubers would normally be riddled and presented on the ware market, whereas crops with damage in excess of this level would usually be used for stock feed or industrial purposes.

One further point must be borne in mind while considering Table 4. In the years during which this work has been carried out it has been found that damage estimates for slug and wireworm are substantially additive. For instance, in 1954 pest damage was found on 447 of the 2,919 fields which were crop checked, but damage by both pests together occurred on only 23 fields. Similarly, in 1955 both pests were found together on 19 out of 276 damaged fields when 2,919 were again surveyed. Table 4 therefore summarizes the net losses due to both pests, an allowance having been made for that part of the total which is common to both species.

In a normal year slug and wireworm together reduce the ware crop in Great Britain by about 65,000 tons, or about 1·3 per cent of production : this is not, however, a complete loss since damaged tubers can be disposed of commercially at a discount of about £3–£5 a ton, which is about 25 per cent of the average ware price. The net annual acreage equivalent loss due to the two pests is therefore of the order of 7,500 in terms of tubers standing on a 1½-inch riddle.

PEST DAMAGE IN RELATION TO SOIL CONDITIONS AND INSECTICIDE USAGE

Information on soil type and cultivations over the three preceding years was collected for each crop visited during the surveys. The main results in relation to soil type are shown in Table 5.

TABLE 5

SOIL TYPE IN RELATION TO PEST DAMAGE, GREAT BRITAIN, 1954–1956

Soil Type	Fields Visited in Three Years		Fields with Wireworm		Fields with Slug	
	Total Number	Total Acreage	Number	Percentage of Total	Number	Percentage of Total
Sand and gravel ..	785	6,994	45	5·7	29	3·7
Silt, skirt, warp ..	1,038	11,592	32	3·1	71	6·8
Peat	553	4,876	26	4·7	21	3·8
Light loam and red ..	1,443	11,396	95	6·6	64	4·4
Medium loam ..	3,512	31,380	192	5·5	240	6·8
Heavy loam	697	6,582	58	8·3	75	10·8
Clay	162	1,914	16	9·9	24	14·8
Limestone	360	3,974	20	5·6	33	9·2
Chalk	77	1,040	3	3·9	3	3·9

There is a slight tendency for both pests to occur on the heavier soils, though it must be stressed that the percentages referring to heavy loam and clay are not founded on really substantial numbers of fields. With slugs, however, there is a trend from the lighter to the heavier soils. Neither pest seems to thrive on the chalk, the 4 per cent of fields infested accounting for 2·7 per cent of the total acreage in both cases.

A summary of pest incidence in relation to potatoes grown on ploughed grassland compared with potatoes grown in arable rotation is given in Table 6. In England and Wales, 12·5 per cent of fields ploughed from pasture showed wireworm damage compared with 6·3 per cent of arable fields, a difference which, in view of the size and nature of the sample, is almost certainly a real one. Slugs, on the other hand, showed little preference for ware on freshly-broken

land, being slightly more troublesome on old arable fields in England and Wales, and considerably more so in Scotland where over 9 per cent of the arable acreage tested was infested compared with 2·5 per cent of the newly-broken land.

TABLE 6

CROPPING IN RELATION TO PEST DAMAGE, GREAT BRITAIN, 1954–1956

Cropping and Locality	Fields Visited in Three Years		Fields with Wireworm		Fields with Slug	
	Total Number	Total Acreage	Number	Percentage of Total	Number	Percentage of Total
England and Wales						
Ploughed from pasture	997	7,516	125	12·5	61	6·1
Arable rotation ..	5,306	49,761	335	6·3	407	7·7
Scotland						
Ploughed from pasture	1,844	17,295	27	1·5	47	2·5
Arable rotation ..	480	5,176	0	0·0	45	9·4

Wireworm damage on potato land can be reduced by the use of insecticides applied by themselves or in a compound fertilizer mixture. In the 1955 survey, information was obtained on this point and it was found that over a third of the ware acreage in the South Eastern Region had received treatment that season, and that about a quarter of the West Midland acreage had been similarly treated. By 1956, insecticide usage had increased substantially, and about 18 per cent of the maincrop fields in England and Wales received treatment in that year compared with 14 per cent in the previous year. As a result of the preliminary information obtained in 1955, Marketing Board inspectors were asked to obtain details of the chemicals applied in 1956, and it was found that aldrin, either as a dust or in a fertilizer mixture, was by far the most popular material. There was no record of *gamma*-BHC being used on any of the crops checked in 1956. The results for both years are summarized in Table 7.

TABLE 7

USE OF INSECTICIDES ON WARE POTATOES FOR WIREWORM CONTROL, 1955–1956

N.A.A.S. Region	Total Number of Fields Tested in Survey		Percentage of Fields Treated		Number of Fields Treated in 1956 with :		
	1955	1956	1955	1956	Aldrin Only	Aldrin Fertilizer	Other Compounds
Eastern	600	562	11	13	21	49	5 (DDT)
South Eastern ..	167	197	38	47	16	76	0
South Western ..	133	121	15	20	8	16	0
West Midland ..	166	175	25	18	12	19	0
East Midland ..	479	492	9	10	10	37	3 (DDT)
Yorks and Lancs ..	325	272	14	26	19	49	3
Northern	172	157	2	13	9	11	1
Wales	95	81	9	7	3	3	0
England and Wales ..	2,137	2,057	14	18	98	260	12
Scotland	782	732	1	2	1	13	0
Great Britain	2,919	2,789	10	14	99	273	12

A mixture of sawdust and turpentine was used to control wireworms in one field in 1956 ; the active ingredient used for the remaining three cases listed under " other compounds " is not known.

It is instructive to compare the information given in Table 7 with the gross wireworm damage figures in Table 2. In 1954, for instance, severe damage occurred to an appreciable extent in the South Western and Yorks and Lancs Regions ; by 1956, 20 and 26 per cent of the fields in these Regions respectively received insecticidal treatment and a marked reduction in severe damage occurred in the latter but not in the former region. In the South Eastern Region no cases of severe damage were noted in 1955 or 1956, possibly consequent on the widespread use of wireworm chemicals. In relation to the corrected net estimates of damage shown in Table 4, however, there seems little enough reason to ascribe the reduction in damage from 1954 to 1956 to an increased use of chemicals. It was not until 1955 that aldrin was widely available for wireworm control in potatoes but the overall loss was greater the following year when appreciably more insecticide was used in many areas ; in fact, in England and Wales 5 per cent of all fields tested in 1955 showed signs of wireworm attack compared with 6 per cent in 1956. The cost of treatment with a proprietary potato wireworm dust is about £4 per acre, a sum which can be recovered if the application enables the grower to put a ton of potatoes on the ware market instead of selling them as sub-standard.

The advisory entomologists in England and Wales carried out potato wireworm control trials with aldrin in 1953 and 1954 (Bevan and Bryden, 1956). The final conclusion from this work was that at population densities below about 500,000 per acre about 4 per cent of tubers may show symptoms of wireworm attack, and occasionally treatment with 2 lb of toxicant per acre is not effective to this extent. In the crop check surveys in 1955 and 1956 visually-assessed wireworm damage amounted to 0·025 tons per acre over 5,026 fields which received no insecticidal treatment, and to 0·013 tons per acre over 682 fields which did receive wireworm treatment. Correcting these estimates to allow for pre-lifting increases in damage, it appears that wireworm treatment reduced damage from about 1·0 to 0·5 cwt per acre on average on the overall 1¼-inch riddle yield of 8·7 tons per acre for the years in question. Clearly, on a commercial scale, treatment must be applied selectively rather than as a routine if it is to be worth while—0·5 cwt of sound ware will scarcely pay for the cost of application, let alone the cost of the chemicals.

Area officers of the Potato Marketing Board and the Potato Division of the Ministry of Food carried out careful gradings and weighings on nearly 9,000 crops in the course of these surveys. Thanks are due to these inspectors, and also to those entomologists in the National Agricultural Advisory Service who co-operated in the lifting checks and clamp examinations.

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SOME OBSERVATIONS ON THE EARLY STAGES OF POTATO BLIGHT ATTACKS

by ANGELA M. CLAYSON AND N. F. ROBERTSON

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Observations in 1955 (Clayson and Robertson, 1956) showed the importance of stem lesions in allowing the survival of *Phytophthora infestans* during July and August in the absence of rain. In continuation of this work, we made observations in 1956 on plants grown in the glasshouse under conditions resembling those in the field plot in the previous summer. We also made observations on the development of blight in a field plot with an artificial source of infection introduced in June; and, by close observation on a commercial crop, we attempted to follow the development of a natural epidemic, and to assess the relative importance of foci arising from infected tubers, spores blown in from other fields, and long-lived stem infections initiated by them.

GLASSHOUSE EXPERIMENT

Thirty-five tubers, variety Eclipse, were planted in a glasshouse on February 7, 1956 and the plants to which they gave rise were inoculated on April 13 with *P. infestans*, Race 4. The inoculations were made by dipping discs of filter paper 6 mm in diameter, or strips of filter paper 3 × 1 cm, in a suspension of zoospores and sporangia and placing them on selected parts of the plants. The largest plants had then eleven leaves and the smallest had just appeared above ground. The inoculation sites chosen were the lamina of the topmost fully-expanded leaf (paper discs), the top internode of the stem, and the stem base. The treatments were randomized over the plot. After inoculation in the late afternoon the plants were left uncovered, but the glasshouse was closed up and all concrete paths were well damped down.

Lesions began to appear on leaves after seven days, on stem tips after ten days and on stem bases after fourteen days. By May 5, twenty-two days after the inoculations, 27 out of 48 inoculated leaves had shown lesions, and most of these had shrivelled or turned yellow and dropped off. There was no spread of infection into the stems. The inoculations of the upper part of the stems gave 31 out of 44 positive infections, and in every instance infection had spread through several internodes. The inoculations at the base of the stems yielded only 6 out of 44 positive infections, mostly confined to one or two internodes.

No overhead watering was given until May 23, by which time all infected leaves had disappeared. The lesions on the stem bases and tips had progressed little, and as the main growing points had grown on for seven or more internodes, the original terminal lesions were now low down on the plants and not obvious on superficial inspection. At this stage the plants were reaching their maximum size and an attempt was made to initiate an epidemic by overhead watering. The plot was watered evenly from above with a spray nozzle for at least 10 minutes every night and morning. In very sunny weather the plants were given a light spraying to wet the foliage at 11 a.m. and again at 2.30 p.m. Maximum ventilation was given in sunny weather to prevent the temperature in the glasshouse becoming too high.

The first sporing lesions were observed on two leaves on May 31, and on June 1 there were more than 60. On June 2, 25 per cent of the shoots showed upwards of 10 lesions each (corresponding to 1 to 5 per cent blight by the B.M.S. scale) and by June 16 the assessment of blight over the whole bed was 95 per cent by this scale, almost all the leaves being destroyed though many stems were still green. Throughout this period there were six more or less continuously cloudy days and the mean minimum temperature in the glasshouse was 53 $\pm$ 1·4° F and the mean maximum 76 $\pm$ 0·8° F. The fungus apparently survived in the stem lesions for at least three weeks before the revival of active sporing by the overhead watering which started on May 23.

FIELD PLOT WITH ARTIFICIAL FOCUS OF INFECTION

The chosen plot was one of King Edward, at the Rockefeller Field Station, Cambridge, and it consisted of 12 rows, each with 22–33 tubers, in the midst of about a quarter of an acre of assorted varieties. The seed tubers were chitted, and planted on April 11, 1956. There was about 25 per cent emergence by May 18, and only occasional shoots were affected by the May frosts. On June 12, when the plants had not yet met in or across the rows, one plant in the centre of the plot was sprayed with a sporangial suspension of Race 4 from the Eclipse in the glasshouse. The plant was covered with an incubation chamber until the afternoon of the following day. Leaf lesions appeared on June 18 and spored on June 20. Stem lesions were well established by July 2. Spread to adjacent plants then followed. By July 6, 28 plants around the infector showed a few lesions ; and by July 14 the number had risen to 109. With Beaumont conditions prevailing from July 12 to 14 and from July 16 to 21, the spread was rapid, and by July 23, 276 of the 348 plants in the plot showed infection ; about one third of them had a few lesions only, and two thirds had many lesions, especially near the ground. The attack on the plot as a whole could then be assessed by the B.M.S. scale, and it amounted to rather over 1 per cent. The subsequent course of the attack is shown in the figure, the 50 per cent stage being reached by August 3 and the 75 per cent stage by August 7.

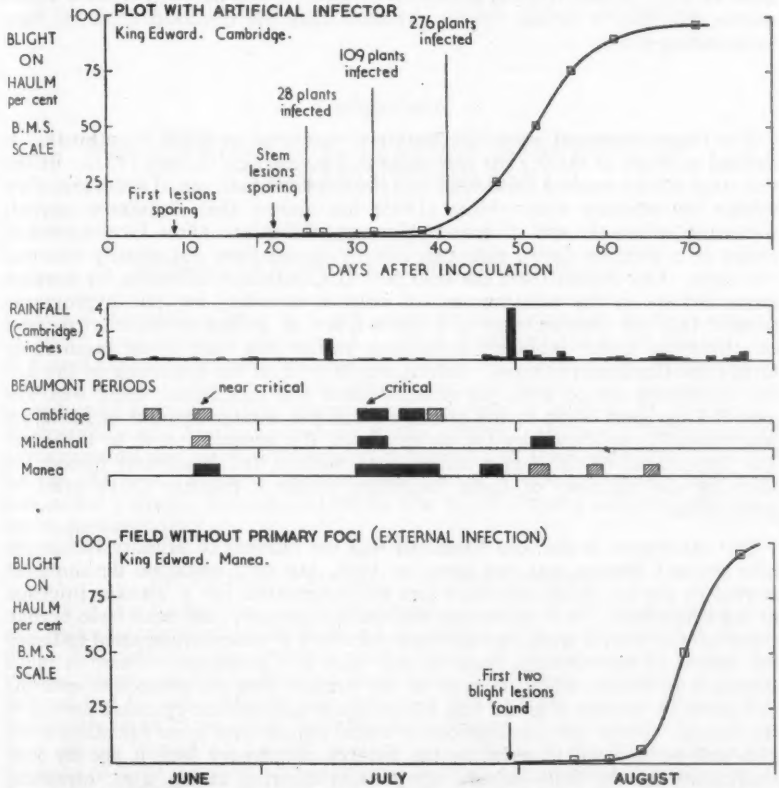
Two recording hair hygrographs were run in this experiment, one in a wooden screen 2 feet above ground level in the crop, and one in a Stevenson screen in the open, 300 yards away. No differences were noted between their recordings. The Beaumont periods and partial Beaumont periods (40–48 hours) recorded are shown in the figure, together with those recorded at the Mildenhall weather station and at Manea, in the Isle of Ely. The rainfall (from records at the Cambridge Botanic Garden, 2½ miles away) is also shown. It will be seen that with the showery conditions prevailing, the disease was able to spread around the artificially inoculated plant before July 14, although on the hair hygrographs no full Beaumont periods were recorded before that date, the humid period on June 22–24 after the first lesions were sporing being of only 40 hours duration.

OBSERVATIONS ON A FIELD CROP

The field selected was at Manea, in the Isle of Ely, a region of intensive potato production and one where blight often starts earlier and progresses more rapidly than at Cambridge. It was a 16-acre field of King Edward, in a central block of which a spraying trial was being conducted by officers of the N.A.A.S., who kindly contributed observations both from their trial plots and from other fields in the district. In addition to the control plots of the trial

area, a central strip about two acres in extent was unsprayed. The part of the field to the east of this strip was dusted with a proprietary dust on July 18 and 23, and the remainder of the field was sprayed with copper oxychloride on July 17 and 24.

It proved impossible to examine every plant in a 16-acre field, and detailed observations were therefore made on 12 rows at the east end of the field with a less detailed inspection of the rest of the field. The rows on which the detailed observations were made were dusted on July 18 and 23. The potatoes were planted on April 16–18, and the first plants were showing above ground on May 16. Search for plants with blighted shoots arising from blighted tubers was rendered difficult owing to frost damage in May, but by June 27 the plants had recovered sufficiently for a thorough examination to be made in the selected rows. No sign of blight was found on this date, nor on July 4, 11, 17, 23 or 31, when the examination was repeated. Two single blight lesions were found high up on a plant in another part of the field on July 30 by the N.A.A.S.



Development of blight in a field plot with an artificial focus started by inoculating a single plant on June 12, compared with that in a field without internal foci of infection. Cambridge and Manea, 1956.

(70903)

officers ; and on August 7 eight infected leaflets were found in several traverses of the field, four of these being in the part closely examined. No blighted shoots such as might have been produced from blighted tubers were seen at any time, and there were no primary foci ; the first infections were widely scattered and only on a few individual leaflets. By August 11 blight was general in the field, about every tenth plant showing a light spotting, and by August 15 nearly every plant showed lesions—about 50 per plant. Thereafter, the attack progressed as shown in the figure, the whole course of the attack being about three weeks later than that in the artificial primary focus at Cambridge.

In the general survey of fields in the neighbourhood, a natural primary focus at Christchurch, 2½ miles away, was found to be well advanced on July 20, and two other probable primary foci were found at March and Chatteris respectively on July 30. During the week August 1–7, blight was found in several fields within a radius of one mile. If it is true, as Van der Zaag (1956) has reported, that sporangia of *P. infestans* can be carried by the wind over a distance of at least 11 km (7 miles) without losing their viability, and as no foci were found within the field, it would appear probable that the infection came in from surrounding fields.

DISCUSSION

For the purposes of economic timing of spraying, a blight " outbreak " is defined as blight at the 0·1 per cent stage in a potato *field* (Large, 1953). Before this stage can be reached there must be a considerable build-up of spore inoculum within the primary foci. Hirst (1955) has shown that Beaumont periods occurring before the end of June in the east of England allow local spread of blight in a primary focus, although distant spread does not usually occur at this time. Our results from the field plot at Cambridge, allowing for possible imperfections in the measurement of relative humidity by hair hygrograph, suggest that the development of a focus from an artificially inoculated plant can progress under humidity conditions rather less than those required to satisfy the Beaumont criteria. Indeed, on July 12, at the beginning of the first full Beaumont period after the infector plant was inoculated, there was well over 0·1 per cent blight in the primary focus and a large amount of inoculum was available, as shown by the sharp rise of the progress curve at this time. The importance of showery weather, even without full Beaumont periods, in allowing the build-up of spore inoculum within a primary focus must be emphasized.

Our experience in the fens suggested that the number of primary foci in the area around Manea was not great in 1956, but that epidemic development started by the few fields with such foci was responsible for a blanket infection of the whole area. It is of interest that the few primary foci must have been in existence for several weeks before their discovery if their development followed the course of the artificial focus in our plot at Cambridge. There is much emphasis in Potato Blight control at the present time on protective spraying and attempts to time it to the best advantage to combat the epidemic spread of the fungus. From our observations it would appear that some attention could also well be devoted to reducing the number of primary foci, if not by their eradication in the field—which, apart from clearing clamp sites, obviously presents great difficulties—at least by attempts to reduce as far as possible the number of infected tubers planted. More survey work on the lines of that carried out in Holland by Van der Zaag (1956) appears to be needed, over a number of years, to reveal how often the first infection in a field does arise from

blighted tubers planted in it, and how often it is started by wind-borne spores from other fields in the locality.

Our thanks are due to Mr. G. H. Brenchley, Mr. E. R. Wallace, Mr. H. A. Wright and Mr. H. W. Wheeler of the N.A.A.S. for their help in contributing observations; to Mr. A. C. Sears for providing facilities for the work in his field at Manea; and to Miss Marion Scott and Mr. J. Bean together with various research students of the Botany School, Cambridge, for field assistance. We are grateful to the Agricultural Research Council for financial help.

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MAYETIOLA DACTYLIDIS KIEFFER IN COCKSFOOT GRASS

by BARBARA M. STOKES

Rothamsted Experimental Station, Harpenden, Herts

Mayetiola dactylidis Kieffer is a gall midge whose larvae live in the stems of cocksfoot grass (*Dactylis glomerata* L.) and is closely related to the Hessian fly, *M. destructor* Say, which occurs on wheat, barley, rye and couch grass (*Agropyron repens* L.). The larvae are distinguishable because those of *M. dactylidis* bear a unidentate sternal spatula whereas this structure is bifid in *M. destructor*. *M. dactylidis* was first described by Kieffer (1896). Houard (1908) reported it from central and western Europe, and Bagnall and Harrison (1918) recorded it from Durham. Puparia received from Dr. H. C. Gough, which had been collected at Feltwell, Norfolk, in 1950 by Mr. A. W. Colling, unfortunately gave rise to parasites only.

COLLECTION OF PUPARIA AND ADULT EMERGENCE

In 1953 this midge was commonly found on cocksfoot at Rothamsted and was collected as puparia from stems in sixteen sites on Rothamsted Farm. Externally, cocksfoot shoots rarely showed signs of infestation, and to find the puparia, shoots, including the stem bases down to soil level, were gathered, and stripped leaf by leaf. The puparia were seen either singly, or, more frequently, in numbers up to 25 per shoot, between the leaf bases. External signs of damage were usually limited to a slight swelling at the base of the shoot due to the presence of large numbers of puparia but without any noticeable thickening of the plant itself. Sometimes the leaves had turned brown though the central leaf might still have been green. Leaf bases inside the shoot were usually browner and frequently moist and rotting. Where the leaf tissue was not rotten the puparia often caused a slight depression where they were lying (Fig. 1).

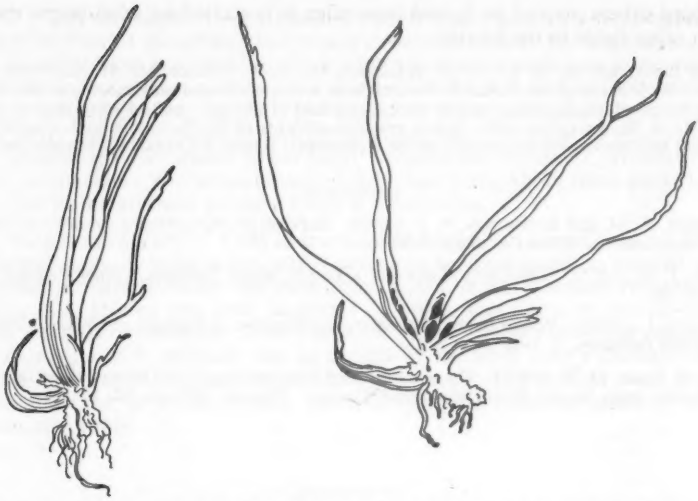

Fig. 1. External appearance of a cocksfoot shoot infested with 25 puparia of *Mayetiola dactylidis*, and (*right*) the shoot opened to show puparia *in situ*.

The puparia were dark, glossy brown and about 4 mm long. Their shape varied slightly when they were numerous and had been compressed in the stem (Fig. 2a). Occasionally paler brown and newly-formed white ones were collected. The larvae inside were typical gall midge larvae each bearing a unidentate sternal spatula which showed some variation in shape (Fig. 2b). The larvae pupate in the stems.

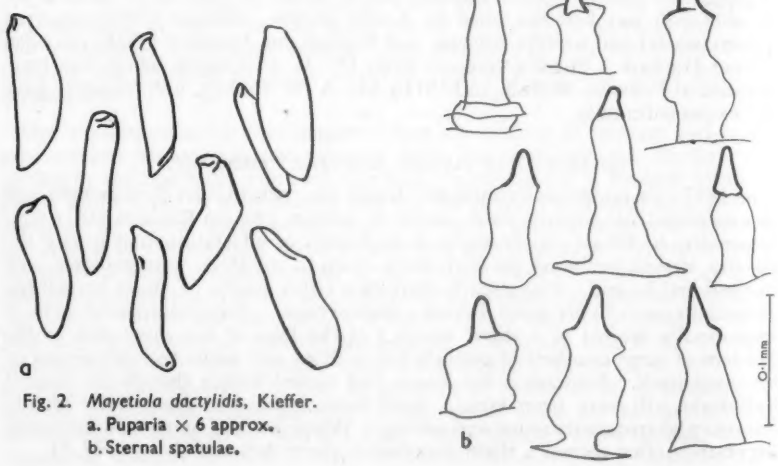

Fig. 2. *Mayetiola dactylidis*, Kieffer.
a. Puparia × 6 approx.
b. Sternal spatulae.

Collected specimens of puparia were placed on damp peat in emergence pots in an open-sided insectary, and the data on collection and emergence of the midges are shown below :

THE COLLECTION OF PUPARIA AND EMERGENCE OF M. DACTYLIDIS

	Puparia Collected	First Emergence	Subsequent Emergences	Max. No. Emerging on Any One Day	Parasites
1953	January 22–April 23	Female May 10	15 males 25 females up to August 29	4 on May 21 4 on June 6 4 on June 11	7 from June 9–July 8
1954	March 2–16	Male May 15	34 males 73 females up to September 21	6 on August 21 6 on August 26 6 on August 27	148 from May 29–September 11
1955	April 15–29	One male and one female June 2	14 males 7 females up to September 24	4 on August 29	No parasites emerged
1956	May 22–June 6	Two males July 19	2 males 3 females up to September 5	2 on July 19 2 on September 5	6 from July 9–25

Although the pots were kept for over a year no midges emerged after August in 1953 or September in the other years. There was no peak period of emergence as the midges came out a few per day.

LIFE HISTORY

Some of the midges were taken from the emergence pots alive and placed on cocksfoot plants grown in 7-inch flower pots, covered by muslin cages, with usually one pair of midges per plant. In all, sixteen attempts were made to breed the midge on cocksfoot. Three of these were successful and a new generation was reared in each case ; in six other pots oviposition was seen and in two further ones larvae and pupae were found although no midges finally emerged.

Observation of the midges showed that mating lasted a few seconds and oviposition began the same day. The yellow-brown eggs, about 0·6 mm long, were laid in groups on the inner side of leaf sheaths at the base of the plant. It was not possible to see when they hatched without disturbing the plants. The parent midges died within a day or two.

Two breeding experiments were begun on June 4, 1954. In one, both brown and newly-formed white puparia were seen on August 13, and additional brown puparia on September 22. The new generation of midges, consisting of no males and 11 females, emerged between May 30 and June 15 the following year. In the other pot, 1 male and 20 female midges appeared between June 2 and September 5, with 4 midges as the highest total on any one day ; none emerged between June 15 and August 27. In the third successful case, egg-laying was seen on August 17, 1954 and puparia on September 22. The new generation of 21 females but no males appeared from August 21 to August 30, 1955, 4 being the highest total on any one day. These three cases suggest that only one generation a year is produced, although midges are active throughout the growing season, and that there is a tendency towards unisexual families bred from individual pairs, as is known to occur in *M. destructor*.

Other grasses which were tested as host plants of *M. dactylidis* by caging pairs of midges with them, were oats (three tests), *Phleum pratense* and *Festuca pratensis* (two tests each), and one each on *Lolium perenne* and *Alopecurus pratensis*. None of these was successful. A female midge which was ovipositing on a caged cocksfoot plant was caught and caged with *Poa pratensis*. It consistently flew off this grass, but, when it was replaced on cocksfoot 1½ hours later, it resumed egg-laying there.

PARASITES

The parasites *Platygaster mayetiolae* Kieffer and *Semiotellus puncticollis* Thoms. emerged from *M. dactylidis* puparia collected in the field. They were kindly identified by Mr. G. E. J. Nixon and Mr. G. J. Kerrich of the British Museum (Natural History) respectively, to whom the author is much indebted. The earliest date on which these parasites appeared was May 29, 1954, and the latest September 11, 1954. *P. mayetiolae* emerged from the puparia collected at Feltwell in 1950.

The author acknowledges with grateful thanks the advice and criticism of Dr. H. F. Barnes, who checked the identification of the midges and in whose midge collection they are available for study. The assistance of Miss Judy Horn and Miss Anne Gray in the collection of puparia is also much appreciated.

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INCREASED POSTAL CHARGES

It is regretted that owing to the increased postage charges, the annual subscription rate for *Plant Pathology* is now 21*s.* 4*d.*; current subscriptions will be subject to the new rates on renewal.

The postage on a single copy of this Journal is 4*d.*

SUGAR BEET YELLOWS IN GREAT BRITAIN, 1956

by R. HULL

Rothamsted Field Station, Dunholme, Lincoln

The incidence of yellows in sugar beet crops was lower in 1956 than in any year in the previous decade. In 149 fields sampled by the agricultural staff of the British Sugar Corporation, an average of 0·05, 0·7, 4·1 and 6·0 per cent of the plants had yellows at the end of June, July, August, and September respectively. Although as many plants as usual had yellows at the end of June, the number increased slowly throughout the summer. The percentage of the entire crop of 408,700 acres with under 1 per cent infection at the end of August was 54·8 ; with 1–20 per cent, 43·2 ; with 21–60 per cent, 1·8 ; and with over 60 per cent, 0·2.

Most of the fields heavily infected at the end of August were near the coast of Suffolk and Essex; the crops with moderate infection were irregularly distributed throughout eastern, central and southern England, and other crops had less than 1 per cent infected plants. At the end of September, the average percentage of infected plants was greatest (11–18 per cent) in the south and in the Ipswich, Bury St. Edmunds, Ely and Cantley factory areas.

The average yield of sugar per acre was the third highest recorded. Yellows caused the following calculated losses in 1956 which were the lowest since these records started in 1946 (c.f. Hull, *Plant Pathology*, 1953, **2,** 39–43 ; 1954, **3,** 130 ; 1955, **4,** 134 ; 1956, **5,** 130) :

Calculated Percentage Loss of Sugar Yield		Actual Yield per Acre		Estimated Potential Yield of Roots per Acre*	Estimated Loss of Root Yield*
Aâ€	Câ€	Sugar	Roots*		
		tons	*tons*	*tons*	*tons (000)*
1·5	3·2	2·05	12·84	13·03	77

* Converted to equivalent yields at a sugar content of 16 per cent.

† A. Assuming 4½ per cent loss for each week the plants show symptons. C. Assuming 2·4 cwt loss for each 10 per cent infected plants at the end of August.

The slow spread of yellows was doubtless the result of the wet, stormy summer restricting the movement and development of aphids ; in the sample fields there were averages of only 1·7 and 6·5 *Myzus persicae* per 20 plants at the end of June and July respectively. In the Holland division of Lincolnshire there was an average of 14·5 green aphids per 20 plants at the end of June, and several growers in this area sprayed their crops with systemic insecticides as a precaution against yellows. N.A.A.S. officers and British Sugar Corporation fieldmen counted plants with yellows in 150 mangold and 107 sugar beet seed crops in June. The results were as follows :

	Percentage of Seed Crops with Yellows				
	under 5%	5–10%	10–20%	20–40%	over 40%
Sugar beet	87	5	3	5	0
Mangold, etc. ..	46	23	22	5	4

Of 137 sugar beet steckling beds examined in October only 2 had more than 1 per cent of plants with yellows and these were destroyed. The average for all beds was 0·10 per cent. Forty out of 344 mangold steckling beds had more than 1 per cent of plants with yellows, the mean for all beds being 0·45 per cent.

A METHOD FOR TRAPPING SAMPLES OF SMALL FLYING INSECTS

by K. S. GEORGE

Plant Pathology Laboratory, Harpenden, Herts

During experimental work on the effects of the Wheat Blossom midges, *Contarinia tritici* Kirby and *Sitodiplosis mosellana* Gehin, on yield of grain, figures indicating the relative abundance of adult midges flying over the crop at different times were required. The apparatus illustrated (Plate I) was designed as an alternative to visual sampling at sundown when the midges are active. It is useful in that it gives a large number of figures for analysis when population density is low, and when the activity periods are so inconvenient as to make other sampling methods tedious.

The trap consists of a bent wire frame, approximately 2½ inches wide and 8 inches long. Three 3 inch by 1 inch microscope slides are held in position by double bends in the wire. In practice, the wire may easily be bent around two nails driven one inch apart into a bench. The wire in the loop formed may be bent so that it forms a spring for holding the slides tightly in position. Finally, the slides are coated on both sides with a suitable adhesive, such as a good proprietary tree-banding compound. Two frames are supported on a wooden cross. The height of the crosspiece, and thus of the frames, above the ground is adjustable so that the traps can be kept at a standard level in relation to the growth of the crop.

In June 1956, four such complete traps were used on each of two unsprayed experimental plots. Catches were examined at the same time as the plots were visually examined for midges. The results were as follows :

	Visual Sampling		Trapping							
	Plot	Plot	Four Traps on Plot A				Four Traps on Plot B			
Date	A	B	U	M	L*	Total	U	M	L	Total
June 11	7	3	0	0	0	0	0	0	0	0
15	3	3	0	0	0	0	0	0	0	0
19	3	3	0	2	3	5	1	0	1	2
20	2	0	1	2	3	6	3	0	0	3
22	0	0	2	2	2	6	1	0	0	1
23	0	0	0	0	0	0	2	0	1	3
24	0	0	1	4	2	7	3	2	5	10
25	0	0	2	3	0	5	1	0	1	2
26	0	0	1	2	2	5	2	3	1	6
27	0	0	2	3	3	8	3	4	3	10

* U, M, L,= upper, middle, and lower, slides.

The trap figures represent midges caught during the preceding 24- or 48-hour period, and the figures obtained from visual sampling represent midges observed during the hour preceding sunset on fifty ears taken at random per plot. There were very few midges to be found in the plot area, and visual sampling clearly did not give a good picture of the position. From the visual data, in fact, the impression might be gained that peak emergence occurred about June 11, whereas the trap figures suggest that it occurred between June 24 and 27.

I am indebted to Mr. A. H. Strickland for the photograph.

Trap erected in a wheat field.

PLATE I

(70903)

REACTIONS OF LUCERNE STRAINS TO VERTICILLIUM WILT (see pp. 137–42)

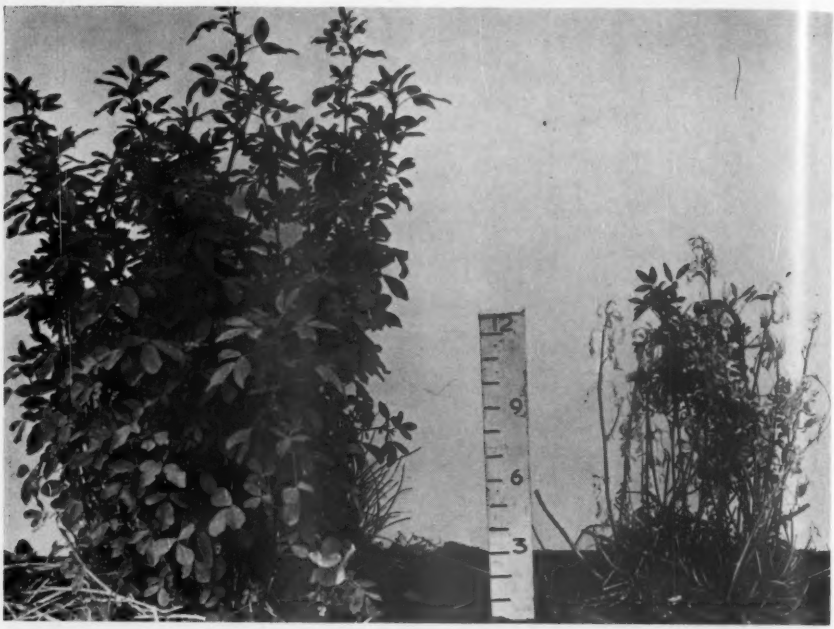
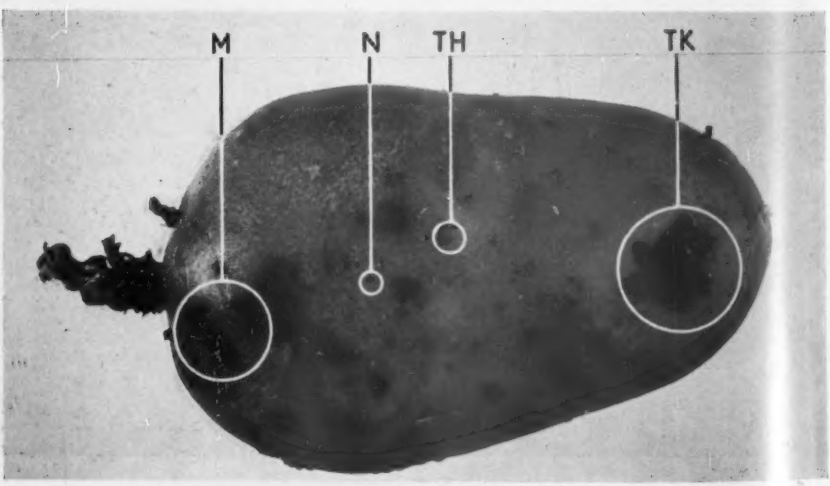


Photo: N.I.A.B.

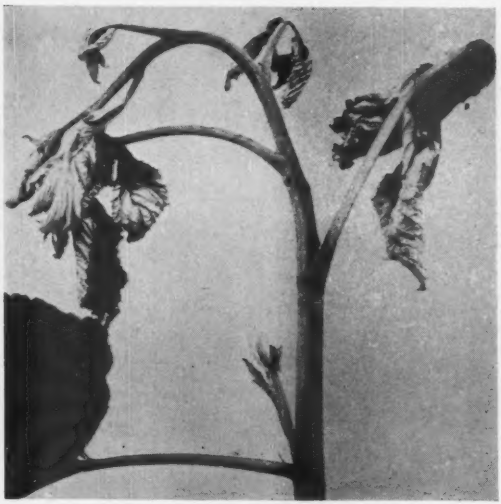
1. *Left*: Healthy plant. *Right*: Plant infected with *Verticillium albo-atrum*.

RHIZOCTONIA SOLANI ON POTATO (see pp. 149–52)



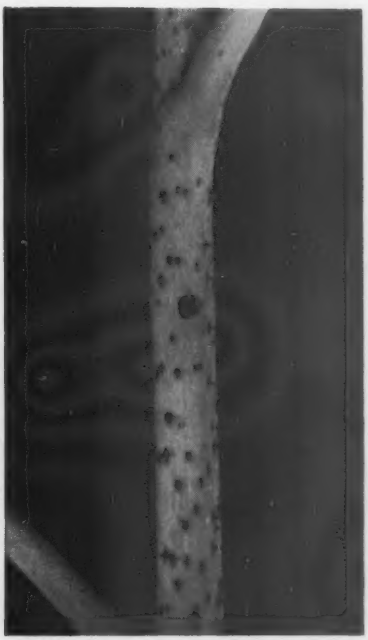
2. Potato tuber showing negligible (N), thin (TH), medium (M) and thick (TK) sclerotia of *Rhizoctonia solani*. ¾ natural size.

DAMAGE TO RASPBERRY BY ROSY RUSTIC MOTH (see *pp. 135–6*)



1. Wilting of apical leaves.

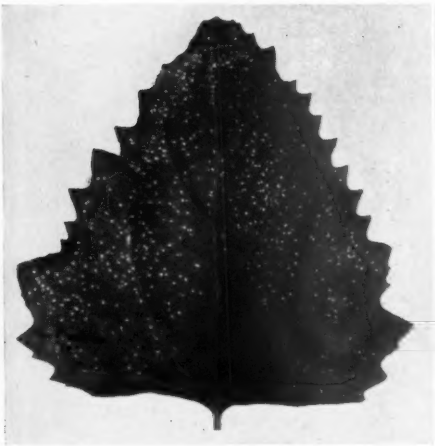
2. Larva in stem cortex.



Photos: North of Scotland College of Agriculture

3. Larval hole in stem.

PLATE III



1. Tobacco necrosis.
Local necrotic dots.

2. Turnip crinkle.
Local chlorotic dots.

3. Potato Aucuba mosaic.
Local necrotic ring spots.

4. Pea mosaic.
Systemic veinal flecks and spots.

PLATE IV

REACTIONS OF SOME ADDITIONAL PLANT VIRUSES ON *CHENOPODIUM AMARANTICOLOR*

by M. HOLLINGS

Plant Pathology Laboratory, Harpenden, Herts

In a previous paper (Hollings, 1956), the reaction of *Chenopodium amaranticolor* Coste & Reyn. to mechanical inoculation with 39 plant viruses was recorded : 24 of these viruses induced symptoms which in many cases were of diagnostic and/or quantitative use.

Further tests by me and by other workers have shown that this plant reacts to a number of additional plant viruses. The methods used in raising and inoculating the plants were the same as in the previous work : M/15 phosphate buffer and " Celite " diatomaceous earth were used in all inoculations. As before, the letter " D " signifies that the symptoms are sufficiently distinctive to be diagnostic, and the letter " Q " indicates that local lesions suitable for quantitative work are produced.

VIRUS SYMPTOMS ON C. AMARANTICOLOR

Anemone brown ring (an undescribed virus from *Anemone coronaria*). Local chlorotic spot lesions 2–2½ mm in diameter after 8–12 days, later enlarging slightly and developing reddish-brown margins. (D, Q)

Barley false stripe (Rothamsted strain). Local yellowish spot lesions 2–3 mm in diameter after about a week, some lesions extending along the veins ; inoculated leaves soon turn yellow and abscise. Kassanis and Slykhuis (unpublished results) had previously obtained this reaction. (D, Q)

Bean yellow mosaic. Few, faint, chlorotic local lesions about ½ mm in diameter after about 2 weeks, later tending to become necrotic. An unreliable reaction, usually occurring only with inoculations from young infected tissues.

Beet mosaic. Bold, semi-necrotic local spots 1 mm in diameter after 5–7 days. Lesions soon enlarge, extending along the veins, with patches of veinal necrosis. Systemic necrotic flecks follow, with patches of net necrosis of smaller veins and considerable leaf buckling. After about a month the younger leaves show only slight or no symptoms. (D, Q)

Broad bean mottle. No reaction obtained with inoculations from infected *Phaseolus vulgaris* L., but from *Vicia faba* L. or *Melilotus alba* Desr. numerous tiny local chlorotic dots develop after about 5 days. Sap from infected *M. alba*, after heating to 80° C for 10 minutes, caused fewer than 20 lesions per inoculated leaf on *Chenopodium*, compared with over 600 lesions per leaf from unheated inoculum. Although the thermal inactivation point of this virus is unusually high (95° C), most infectivity would seem to be lost at lower temperatures.

Chrysanthemum latent. (Hollings, 1958). Numerous tiny local yellow-green dot lesions after 4–6 days. (Q)

Hydrangea ring spot. Numerous chlorotic dot local lesions, each with a central necrotic speck, after 5–7 days. (Q). Previously recorded by Brierley and Lorentz (1956).

Lettuce mosaic. When previously tested, no reaction was obtained in several attempts from a large, infected lettuce plant. But when inoculations were made from young, infected lettuce seedlings, a distinctive reaction was obtained. Bright yellow-green local spot lesions developed after 6–12 days ; the lesions enlarged and, 1–2 weeks later, systemic yellowish spots and veinal flecks appeared on the younger leaves, with variable leaf buckling and distortion. After several weeks, young leaves are produced with much milder symptoms. (D, Q)

Lucerne mosaic. Numerous, rather faint semi-necrotic local dot lesions after 4–6 days. Rapid systemic invasion a day or two later, with chlorotic flecks, streaks and spots, severe leaf curling and buckling on the young leaves, which have a grey, mealy appearance. Plants severely stunted. (D)

Narcissus stripe. As previously reported, no reaction was obtained in inoculations from leaves of narcissus plants showing stripe symptoms. Inoculations from flowers of 8 such plants, however, caused irregular, diffuse, rather faint chlorotic local spots 2–4 mm in diameter after about a week. From these lesions the virus was readily transmitted to *Gomphrena globosa* L., and back to *Chenopodium.* There is, however, no evidence that this virus is the cause of stripe symptoms in daffodil.

Pea mosaic. Local chlorotic spot lesions about 1½ mm in diameter after 6–14 days. After a further 3–15 days systemic chlorotic spots and veinal flecks develop, intensifying into a brilliant, coarse yellow-netting of the veins. (Plate IV, 4) (D, Q)

Potato virus G (*Aucuba mosaic*). Few, whitish semi-necrotic local rings, ring spots and concentric rings after 5–8 days, later extending to ring and line patterns. (Plate IV, 3) (D)

Tobacco necrosis. Five isolates. Numerous pale fawn, necrotic local dots after 2–5 days ; lesions from some isolates develop into brown necrotic rings, others remain as pale dots. (Plate IV, 1) (D, Q)

Tobacco ring spot. Two strains previously tested gave local lesions only, but an isolate kindly supplied by Dr. J. H. Hitchborn, Virus Research Unit, Cambridge, produced local necrotic dots after about a week, and a few days later caused systemic chlorotic spots and veinal flecks, with considerable leaf buckling.

Tomato black fleck (an undescribed virus from tomato, serologically related to tobacco mosaic, but differing from all other strains of TMV tested in its symptoms on tomato, and on *C. amaranticolor*). Numerous faint, semi-necrotic local lesions of irregular size and shape, after 3–5 days ; systemic symptoms after a further 1–2 weeks, with yellowish-grey veinal flecks and spots which become necrotic, causing considerable leaf buckling and curling. Growth severely checked, and plants sometimes killed in winter. (D)

Turnip crinkle. Numerous local chlorotic dots after 5–7 days, the lesions remaining discrete. Previously reported by Broadbent and Heathcote (1956). (Plate IV, 2) (Q)

No reaction could be detected after inoculation with any of the following viruses : carnation latent, chrysanthemum ring pattern, chrysanthemum vein mottle, potato veinal necrosis (= tobacco veinal necrosis), and one isolate of potato virus C. A previous isolate of potato virus C induced numerous local chlorotic dot lesions after about a week, these lesions being indistinguishable from those of potato virus Y. Another isolate of virus C, kindly supplied by

Dr. Kassanis of Rothamsted, caused very few local lesions, which enlarged to about 2 mm in diameter and developed brown necrotic margins. Potato veinal necrosis, potato virus C and potato virus Y are serologically related, and it may be that there are, within this group, viruses with properties intermediate between those of typical members.

As reported in the previous paper, *C. amaranticolor* reacts with diagnostic local lesions to inoculation with carnation ring spot, carnation mottle and carnation vein mottle viruses. A rapid method of indexing carnations for these viruses was developed, using a modification of Yarwood's dry-inoculation technique. The tip of a carnation shoot is cut vertically into two with a safety-razor blade, and the cut surfaces gently wiped on leaves of *C. amaranticolor*, previously dusted lightly with 400-mesh carborundum powder. If more convenient, an alternative method is to scrape the epidermis from a young carnation leaf, and wipe the cut surface over the *Chenopodium* leaves. In either method, the result is usually visible within a few days, and the test has been found much quicker and more sensitive than the conventional one using *Dianthus barbatus* or similar species.

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DAMAGE BY ROSY RUSTIC MOTH LARVAE IN SCOTLAND, 1956

by M. W. SHAW

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Caterpillars of the Rosy Rustic moth (*Gortyna micacea* Esp.) are found in the field in most years but rarely cause damage of economic importance. This seems true of countries other than Great Britain where occasionally serious infestations occur, such as those recorded on rhubarb in Nova Scotia (Brittain, *Rep. N.S. Dep. Agric.*, 1915, 28–51 ; 1924, 50–9) and on onions in Russia (Esterberg, *Pl. Prot., Leningr.*, 1932, **2**, 79–82). During 1956, cultivated crops throughout Scotland, particularly raspberry, iris, rhubarb and potato, suffered severe damage.

Outbreaks in raspberry crops seemed to be confined to eastern Scotland, from Midlothian to southern Kincardineshire. Although Dr. A. R. Hill (personal communication) received typically damaged canes from a Dundee grower in

1947, damage to this crop has previously been recorded only from Sweden (Tullgren, *Medd. CentAnst. Försöksv. Jordbr., Stockh.*, **152** ; *Entomologiska Avdelningen*, **27**, 1–104). In a field at Muirhead, Angus, some 5 per cent of the young canes were infested, and about 50 per cent in a cane nursery near Dundee. Similar damage was found in a fruiting plantation at Marykirk, Kincardineshire, and in other areas of eastern Scotland.

The first symptom of attack was wilting of the apical leaves (Plate III, 1). On splitting the stem, a young larva was usually found mining the cortex (Plate III, 2)—a characteristic of the species on other hosts. Holes made by the larva entering or leaving the stem (Plate III, 3) could be found anywhere from the base to within a few inches of the growing point. In some cases damaged stems were found without a caterpillar, but mining was confined to just underneath the epidermis, after which the larva had retraced its path and vacated the stem. As internal damage increased, the leaves became dry and brittle, and eventually died.

Iris crops in Britain are rarely attacked but injury was recorded from Angus in 1950 (*Rep. Edinb. Coll. Agric.*, 1950, 67) and found in Aberdeenshire in 1954. Van Poeteren (*Versl. PlZiekt. Dienst Wageningen*, **44**, 124) has reported damage in Holland. A serious outbreak occurred in a bed of 2,500 iris bulbs in south-east Scotland where 90 per cent of the plants were severely damaged and rendered useless, the bulbs and young shoots being extensively mined. Larvae of different instars were found both in attacked raspberry and iris plants and all instances of damage to both crops were reported between June 15 and 22.

Serious injury to rhubarb appears to be rare. Cameron records its occurrence in Scotland (*Trans. R. Highl. agric. Soc. Scot.* 1938, **50**, 109), and records at the Plant Pathology Laboratory, Harpenden, show that the late G. Fox Wilson reported damage from London allotments in 1944. Brittain, as already stated, records severe damage in Nova Scotia, and Vappula (*Puutarha*, **35**, 3, 69–71) mentions similar injury in Finland. Crops at Kirriemuir, Balgillo, and Dundee, Angus, where 75 per cent of the stalks were attacked, were rendered unsaleable through internal feeding tracks and " bleeding " from the wounds made by the larvae.

Damage to potatoes was common throughout the country, being particularly severe in gardens and allotments in Kirkcudbrightshire, Ayrshire and west Perthshire. In south Kincardineshire the attack on seed crops was sufficiently intense to be noticed by the growers. Strawberries, sugar beet and gladioli in the east and south-east areas were also attacked.

From the annual reports of the Scottish Agricultural Colleges, it appears that this insect was last abundant in Scotland in 1952, but in most years populations are too small to cause damage of economic importance.

I wish to thank Dr. D. S. MacLagan, Mr. E. Dunn and Mr. A. G. Fisken for contributing records without which this note could not have been prepared.

REACTIONS OF LUCERNE STRAINS TO VERTICILLIUM WILT

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In the autumn of 1952 an intensive wilting, yellowing and dying of leaves and stems was observed on a few of the single-spaced plants grown at the N.I.A.B. The disease was identified and the fungus was isolated at the Botany School, University of Cambridge, as *Verticillium albo-atrum* Reinke & Berth. This disease in lucerne in Britain had been reported and described by Mary Noble *et al.* (1953). Since 1952 the disease has occurred each year in East Anglia and recent reports indicate its presence in the north and west. The presence of wilt at the N.I.A.B. enabled studies to be made on the relative resistance of lucerne strains to the disease.

FIELD OBSERVATIONS ON SINGLE-SPACED STRAINS

Detailed observations were made in 1953 and 1954, after the disease was confirmed, on the twelve strains (*see* Tables 1 and 2) of lucerne planted, for identification purposes, in the spring of 1951 on heavy, fertile soil. Two hundred plants of each strain were planted out 18 inches apart each way. They were in four blocks, with 50 plants of each strain to a block, but two year's observations were confined to three blocks. When in 1953 records of a number of diseased plants were taken, some plants were dead in each strain as the stand was then two years old.

Observations were made each month on individual plants for apparent disease symptoms as illustrated in Plate II, 1. On the evidence of these symptoms they were recorded as infected plants. All strains were cut on the same day three times a year. After cutting, the plants seemed to recover, especially in quick-growing early strains, cf. Zaleski (1954). They showed very few symptoms of disease in the aftermath (2–3 weeks after cutting) but the symptoms re-occurred again when the plants reached the flower stage.

The plants showed the most severe symptoms of wilt towards the end of the summer and during the autumn of 1954. The fungus had probably ample opportunity to spread extensively by that time. The progress of infection and mortality among the strains are shown in Table 1.

From this table a few interesting points have arisen. There were differences between strains in both percentage of infection and mortality. The death of plants was negligible during 1953 (from May until October). The percentage of infected plants in May 1953, October 1953 and May 1954, showed little variation, the percentage during the last period being slightly lower than in the two earlier periods. The mortality was of some significance by spring 1954. It was the highest in Hunter River and Saladina, which are not winter-hardy strains, as shown by Zaleski (1954). These two strains lost a considerably lower proportion of plants than others during summer 1954 (from May till October). In most of the remaining ten strains the heaviest losses occurred not during the winter 1953/54 but during the summer of 1954. The percentage of infection in May and October was greatest in Chartrainvilliers and W.268 strains, but by May 1954 the difference between strains was not significant. The highest infection

was recorded in the middle of November each year, but these records were thought to be unreliable as towards the autumn plants are generally " dying off ", some strains reaching the dormancy stage earlier than others, and often the natural dying off associated with dormancy can be mistaken for disease symptoms.

For this reason, in November 1953, shoots from nine plants which appeared to be infected, and nine shoots from plants which appeared to be healthy were chosen at random from each replicate of Du Puits, Chartrainvilliers and Strain B and examined for presence of the fungus. It must be emphasized that Strain B reached dormancy earlier than Du Puits or Chartrainvilliers and it was difficult to decide at that time which plants were healthy or infected. Verticillium Wilt was confirmed by petri-dish tests in all plants of Du Puits and Chartrainvilliers recorded as infected, but some plants of Strain B, which were recorded as diseased, failed to show presence of the fungus. On the other hand, it was present in some of the plants recorded as healthy. This indicates that at the time of year when some plants reach dormancy stage, the disease symptoms can be mistaken, and therefore the November figures were not considered to be reliable.

TABLE 1

PERCENTAGES OF INFECTED AND DEAD PLANTS

Type and Strain	Total Number of Plants Alive (T) and Percentage Infected (I%)								Percentage of Plants Dying* During Period	
	1953				1954				May 1953 to May 1954	May 1954 to Oct. 1954
	May 20		Oct. 6		May 26		Oct. 5			
	T	I%	T	I%	T	I%	T	I%		
Early										
Du Puits	120	16	119	17	100	16	80	45	16	20
Eynsford ..	129	15	127	14	115	15	84	45	11	27
Chartrainvilliers	129	29	124	29	105	19	80	55	18	24
W.268 ..	130	29	123	31	99	20	78	49	24	21
Mid-season ..										
Provence ..	143	16	137	17	128	18	99	40	10	23
Marlborough	138	19	132	18	120	20	90	40	13	25
Strain B ..	126	15	121	16	106	16	74	42	16	30
Semi-late										
No. 1 ..	127	23	123	22	105	20	73	33	17	30
Hunter River	90	21	90	21	62	18	52	38	31	16
Late										
Grimm ..	131	17	128	14	95	14	70	45	27	26
Rhizoma ..	134	14	128	17	102	15	66	38	24	35
Extra-late ..										
Saladina ..	68	16	60	20	36	16	29	41	47	19

* Expressed as a percentage of number of plants alive at beginning of corresponding period.

To compare the percentage mortality in plants which were infected or healthy in spring 1953 (*see* Plate II, 1), the plants were marked and the number of dead plants was recorded in May 1954 (Table 2). In general, a number of factors affect the persistency of plants, but the extremely high proportion of death in infected plants as compared with that in healthy plants indicates that in all strains the main cause of mortality was the fungus. This is quite apparent when

the percentage of death in healthy plants is deducted from the percentage of death in infected plants in each strain (*see* last column of Table 2). The high mortality among plants of Hunter River and Saladina strains was caused partly by their lack of winter hardiness, but it is interesting to note that Grimm and Rhizoma, late winter-hardy strains, also showed significantly higher mortality than other strains. By the autumn of 1953 the trial became contaminated by weeds. This affected all strains, but a number of investigations have shown that late strains when grown under comparable conditions are more sensitive to weed competition than early or mid-season strains.

TABLE 2

NUMBER AND PERCENTAGE MORTALITY IN HEALTHY AND INFECTED PLANTS

Type and Strain	Plants Infected in Spring 1953				Plants Healthy in Spring 1953				Estimated Percentage of Infected Plants Killed by the Fungus
	Total No.	Dead in Spring 1954 No.	%	A*	Total No.	Dead in Spring 1954 No.	%	A*	
Early									
Du Puits ..	19	11	58	49·6	101	9	9	17·5	49
Eynsford ..	20	8	40	39·2	109	6	6	14·2	34
Chartrainvilliers	37	16	43	41·0	92	8	9	17·5	34
W.268	38	22	58	49·6	92	9	9	17·5	49
Mid-season									
Provence ..	23	7	30	33·2	120	8	6	14·2	24
Marlborough ..	27	10	37	37·5	111	8	7	15·3	30
Strain B ..	19	8	42	40·4	107	12	11	19·4	31
Semi-late									
No. 1	29	15	51	45·6	98	7	7	15·3	44
Hunter River ..	20	12	60	50·8	70	16	22	28·0	38
Late									
Grimm.. ..	23	16	69	56·2	108	20	18	25·1	51
Rhizoma ..	19	15	79	62·7	115	17	15	22·8	64
Extra-late									
Saladina ..	11	8	72	58·1	57	14	24	29·3	48
S.E.				± 5·85				± 2·87	
S.D. (P = 0·05)				17·0				8·0	
S.D. (P = 0·01)				23·0				10·5	

* A=percentage after angular transformation.

FIELD OBSERVATION ON STRAINS IN ROWS

In the autumn of 1953, one of the four blocks with single-spaced plants was ploughed up ; many of the plants within it showed the disease symptoms and it could be assumed that after cultivation the soil was fairly evenly contaminated. Six strains (Table 3) were drilled in spring 1954, at 4 lb per acre in rows 18 inches apart in ten randomized blocks, especially for observation on their resistance to the fungus. The land was rather dirty but the establishment of plants was fairly even in rows for each strain and there was little or no sign of disease in the year of sowing.

To ascertain the relative differences between these strains in the degree of infection during 1955 and 1956, the disease was recorded by two methods : by monthly estimation of the degree of infection on a comparative basis from 0 to 10

(0 = healthy plants ; 10 = most of plants infected) and by three estimations (each approximately a week before defoliation) of the number of healthy and diseased plants for each strain. All strains were cut on the same day three times a year.

The June estimation was two weeks after the first cut, and the August estimation two weeks after the second cut. The symptoms of disease were less severe and fewer plants were infected in 1955 than in 1956. Table 3 gives the degree of wilt attack in the second harvest year estimated by two methods expressed as a mean for each strain.

TABLE 3

DEGREE OF INFECTION ON LUCERNE IN ROWS, 1956

Type and Strain	Infection on 0 to 10 Scale					Percentage of Infected Plants			Percentage of Dead Plants*
	May	June	July	August	Sept.	May	July	Sept.	Oct. 1956
Early									
Eynsford ..	4·2	3·0	6·3	3·4	7·2	29	54	68	59
Du Puits ..	4·0	3·1	5·9	3·2	7·3	29	53	70	58
Chartrainvilliers	5·3	3·3	6·4	3·7	7·5	31	59	73	62
Mid-season									
Provence ..	4·4	3·3	6·2	3·4	6·6	30	61	66	57
Strain B ..	5·8	3·9	6·3	3·8	7·7	49	65	78	64
Late									
Grimm.. ..	3·4	4·2	5·6	4·6	6·3	28	60	64	76

* Based on estimated number in October 1955.

Both estimations confirmed that the worst symptoms of disease are apparent towards the end of the season when rate of lucerne growth usually slows down and, owing to the age of plants, the fungus can spread and produce symptoms more readily. They also indicated that after cutting, the earlier strains, which perhaps owing to their more advanced stage of maturity usually showed worse infection at the time of cutting than the later strains, seemed to recover much better from the symptoms (probably owing to their vigour of growth) grew quickly away from the fungus, and sustained less damage than a late strain.

There was no great variation between strains in the severity of disease, but again the high mortality in Grimm strain was rather outstanding. The trial was contaminated with weed, and as Grimm lucerne is slow in spring growth and in recovery after cutting and also becomes dormant early in the season, it suffered more severe infestation by weed than other strains. This combined with fungus damage might cause the comparatively heavy loss of plants.

GLASSHOUSE EXPERIMENT

A test on the relative resistance of lucerne strains to Verticillium Wilt was carried out in a glasshouse. Five strains (Table 4) were sown in May 1955 in boxes containing sterilized potting compost. There were five boxes for each strain. In four out of each set of five boxes 200 g of corn meal sand agar containing *Verticillium albo-atrum* was shaken over each box, and lightly covered with sifted soil. Fifteen plants, spaced 2 inches by 3 inches were allowed to grow in each box, and one box per strain was used as a control.

By the end of June plants had reached the flower bud stage and, with the exception of one plant in Du Puits, two plants in Provence and one plant in Grimm, there were no wilt symptoms. All the plants were cut and further inoculum was placed round the crown of each plant in the infected boxes. By the end of August the symptoms of disease were apparent in all inoculated strains but not in uninoculated control strains. Only stunted plants with some leaves and shoots partly desiccated were recorded as diseased. There were a certain number of plants in infected and control boxes showing discoloration of leaves, but this was thought to be due rather to starvation effects as the same symptoms were apparent in about 1,000 plants of lucerne grown in boxes at the same time for other purposes. After recording, all strains were cut according to maturity stage.

TABLE 4

PERCENTAGE OF INFECTED AND DEAD PLANTS IN STRAINS INOCULATED WITH THE FUNGUS

Type and Strain	Percentage of Infected (I) and Dead (D) Plants in Glasshouse, 1955						Percentage of Infected (I) and Dead (D) Plants in Field, 1956											
	August		Oct.		Controls Oct.		May		June		July		Sept.		Controls Sept.			
	I	D	I	D	I	D	I	D	I	D	I	D	I	D	I	D		
Early																		
Eynsford ..	23	4	40	14	0	1	82	15	75	15	90	40	100	60	80	28		
Du Puits ..	51	4	62	17	0	0	89	30	78	40	90	55	100	65	76	25		
Mid-season																		
Provence ..	52	5	50	17	0	0	85	14	82	17	85	52	100	59	70	20		
Strain B ..	53	5	61	18	0	0	94	29	84	29	93	45	100	62	81	30		
Late																		
Grimm ..	45	5	67	17	0	1	60	40	57	40	91	51	100	64	70	29		

After cutting, the recovering plants at first showed little or no sign of disease but soon the process repeated itself. The disease was recorded again in October (*see* Table 4) and afterwards strains were cut according to stage of maturity. By the end of October, both inoculated and control plants with the soil from the boxes were transplanted outside into contaminated soil and the same distance of 2 inches by 3 inches between plants was kept.

In 1956, transplanted plants of all strains were cut on the same day at the end of May, beginning of July, and September. The Eynsford strain, which showed a comparatively low degree of infection in the glasshouse, after transplanting outside reached a similar degree of infection to the others by May 1956. Grimm in May and June had a lower percentage of diseased plants outside, probably owing to a less advanced stage of growth. By July, the disease had spread in the closely-planted plants and symptoms were apparent in all strains to about the same extent. It is worth noting that the percentage of actual dead plants in the glasshouse in 1955 and outside in September 1956 is very similar for all strains when there was no weed competition. This lends support to the theory that the high mortality in Grimm when grown in the field as single-spaced plants or in rows was due partly to weed competition.

The control plants (Table 4), which showed no apparent disease symptoms in the glasshouse, when transplanted on the contaminated soil showed a high degree of infection by September 1956.

It is difficult to explain how the fungus could first have been introduced into the soil at the N.I.A.B. As far as records go, no lucerne crop has been grown previously on this piece of land. It has been alternately under grasses and red or white clovers for the last twenty years. The most reasonable explanation is that the fungus was introduced with one or more strains of lucerne seed, and that subsequent spread of the disease took place after the lucerne was planted in 1951.

Some two hundred yards away from the area known to be contaminated with Verticillium, another area had been sown for the first time with lucerne in the spring of 1954. Some 80 strains were represented in this observation trial. By 1956 Verticillium was apparent in most of these strains. As lucerne has not previously been grown on this land the disease presence in 1956 can either be attributed to seed or, more likely, to the transfer of the fungus from the contaminated area by the same implements which had been used constantly for various cultivations in both areas.

CONCLUSIONS

Verticillium Wilt attacked all strains used in these investigations, but there was a certain relative difference between strains in the degree of infection. The symptoms were more severe towards the end of the season and the disease spread with increasing age of the stand. Loss of plant was greatest in the late strains, where damage due to weed competition was contributory. The disease symptoms were more pronounced in early strains at the time of defoliation, probably owing to their earliness in maturity, but they recovered more quickly after cutting and sustained fewer casualties than late strains. Provence also had lower mortality than most other strains. The damage of the disease to the crop was caused not only by the mortality of plants, but also by the wilting and desiccation of the foliage which naturally would affect both the quality and quantity of fodder. This damage can be reduced by proper management. As the severe wilting and dying of foliage takes place in a diseased crop when it reaches the full maturity stage, lucerne should be cut early (at the flower bud stage) so that the fungus will have less opportunity to cause much loss.

The hope of finding a completely resistant strain among those strains commonly grown in this country seems to be very remote and it may be necessary for breeders in this country and in Europe to select or breed the strains which would be resistant to this disease. Meanwhile, a more intensive research into the whole problem of Verticillium Wilt, including its control, is needed.

Thanks are due to Dr. N. F. Robertson of the Botany School, Cambridge, for identification and isolation of the fungus; to Dr. I. F. Storey of the N.A.A.S. for arranging the petri dish test; and to Miss J. Ives of the N.A.A.S., Cambridge for helping with the glasshouse test.

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FRIT FLY AND THE OAT PANICLE

II. DAMAGED GRAIN

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In the first part of this contribution (Empson, 1957), it was suggested that the weight of a healthy oat grain depends on three factors: its position in the spikelet, the position of that spikelet within the panicle, and a factor which is common to all the grains in a panicle but which is not related directly to panicle size. The present article considers the influence of Frit attack in one grain on the weight of a healthy grain in the same spikelet, the distribution of attack within and between panicles, the weight and kernel content of fritted grain, and the effect of Frit attack on the chemical composition of the grain. The paper ends with a note on the application of the results to the assessment of damage by the summer generation of the Frit fly. Definitions of the terms used to describe parts of the panicle and conditions of the grain were given in the first paper.

FRIT ATTACK WITHIN SPIKELETS

To study the influence of Frit attack in one grain on the weight of a healthy grain in the same two-grained spikelet, it was necessary, because of weight variation within and between panicles, to take spikelets in pairs. One of each pair of spikelets had both grains healthy whereas the other had only either the main or the bosom grain attacked. The pairs were taken from similar positions in each of twelve panicles and the following average kernel weights were obtained:

Mean weight (mg) of 24 main kernels when bosom grain was :	healthy ..	32·5
	fritted ..	31·5
Mean weight (mg) of 63 bosom kernels when main grain was :	healthy ..	20·7
	fritted ..	20·1

Main grains suffer a greater degree of attack than bosom grains (Cunliffe and Fryer, 1924), and there were comparatively few spikelets in which the bosom grain was attacked and the main healthy. Spikelets with healthy bosom grains from a crop of Onward grown in 1953 were divided into groups according to whether the associated main grain was healthy, fritted or blind. The results are given in Table I, and it must be emphasized that, although they are in general agreement with those set out above, they could be biased by an uneven intensity of attack within and between panicles.

TABLE 1

AVERAGE WEIGHTS (MG) OF HEALTHY BOSOM GRAINS

Associated Main Grain	Number Weighed	Husk	Kernel	Whole Grain	Percentage Kernel
Healthy	6,994	6·6	20·8	27·4	75·9
Fritted	525	6·4	20·1	26·5	75·9
Blind	134	6·8	23·2	30·0	77·3

It seems, from these results, that there is little, if any, compensatory increase in the weight of healthy grains adjacent to attacked ones; in fact, the weight of

such grains in both husk and kernel is slightly decreased. However, where the main grain is blind, the adjacent bosom grain is increased in both weight and kernel content.

ATTACK WITHIN AND BETWEEN PANICLES

Examinations were made of a series of panicle samples from crops with widely differing levels of grain attack to analyse the distribution of damage within and between panicles. The results are shown in Table 2.

TABLE 2

DISTRIBUTION OF DAMAGE WITHIN PANICLES

Percentage Attack (All Grains)	Number of Panicles Examined	Percentage of Spikelets with Attack on: Main Grains (M)	Bosom Grains (B)	Both Grains *Observed*	Both Grains *Expected* (M × B/100)	Differences between *Observed* and *Expected* in Angular Transform	Ratio M/B
73·5	10	83·7	63·3	56·3	53·0	1·9	1·3
44·9	10	55·1	34·6	22·8	19·1	2·7	1·6
37·0	25	56·5	17·5	13·5	9·9	3·3	3·2
32·2	25	47·6	16·8	10·7	8·0	2·7	2·8
29·3	25	45·9	12·7	8·1	5·8	2·6	3·6
20·9	10	32·0	9·8	3·1	3·1	0·0	3·3
18·7	25	28·5	8·9	6·1	2·5	5·2	3·2
17·6	25	29·3	6·0	3·5	1·7	3·1	4·9
10·6	25	16·7	4·4	1·9	0·7	3·1	3·8
8·1	25	13·5	2·8	1·3	0·4	2·9	4·8
4·1	25	7·1	1·2	0·2	0·1	0·8	5·9
2·0	10	3·7	0·3	0·0	0·0	−0·6	12·3

Main grains tend to be attacked to a greater extent than bosom grains, a fact noted by Cunliffe and Fryer (1924) over a series of twelve oat varieties. This effect is not uniform over all levels of attack however, tending towards unity at high, and a ratio of about 4 to 1 at low, larval densities. The ratio of 12·3 to 1 in the bottom line of the table may result from error due to the small size of the sample examined—in another series of counts 1,000 grains were dissected from each of 56 samples (from 28 crops) in which less than 10 per cent of the grains were fritted ; the main/bosom ratio was found to be 4·1 to 1.

When the percentage attack on both classes of grain is known, it is easy to calculate the proportion of spikelets likely to have both grains fritted if it is assumed that attack falls evenly on all spikelets. The figures obtained using the formula (M × B/100) are shown in the *Expected* column of Table 2 for comparison with the observed results obtained by dissection. The observed values are appreciably greater than those expected if an attack were evenly distributed—the mean angular difference is 2·31 ± 0·457—and it is clear that an attack does not fall at random on all the florets of each class. This is not surprising because, in any given crop, there are always some florets or even whole panicles which are unsuitable for oviposition or larval development. There is no readily detectable relationship between these differences and the degree of attack, and no satisfactory explanation can be advanced to explain their variation.

The degree of Frit attack was investigated in relation to the position of the spikelets in the panicle, and also in relation to panicle size, by dissecting eight samples, each of 25 panicles, including the varieties Blenda, Sun II and Star. The results are shown in Table 3.

TABLE 3

MEAN PERCENTAGE FRIT ATTACK IN ANGULAR TRANSFORMATION

Whorl Number	Large Panicles Main	Large Panicles Bosom	Small Panicles Main	Small Panicles Bosom	Mean Attack per Whorl (±1·35)
1	22·8	6·5	28·0	13·7	17·8
2	29·7	9·5	27·4	16·2	20·7
3	28·7	15·3	35·0	10·3	22·3
4	31·5	13·0	32·5	15·2	23·0
5	29·6	15·0	36·8	17·7	24·8
6	31·3	17·5	39·9	16·1	26·2
(7*)	(37·8)	(17·4)	(31·9)	(18·4)	(26·7)
Means (1–6)	28·9	12·8	33·3	14·9	22·5

Mean for panicle size : large 20·9 small 24·1 (±2·26)

Mean for type of grain : main 31·1 bosom 13·8 (±0·78)

* Owing to the high proportion of singles and sterile spikelets in the seventh whorl in several of the panicles, these data were excluded from the analysis from which the standard errors were calculated.

Infestation increases towards the base of the panicle in both classes of grain. Main grains clearly suffer a greater degree of attack than bosom grains throughout the panicle, but a detailed analysis of variance shows that there is no evidence of differences in the main/bosom attack ratio in different parts of the panicle. There is some slight evidence (P = about 0·08) that the difference in infestation between large and small panicles may not be altogether a chance effect.

WEIGHT AND KERNEL CONTENT OF FRITTED GRAIN

The mean weights of husk, kernel, and whole grain, together with the figure for kernel content, for fritted grains of each class are given in Table 4. If these are compared with the figures for healthy grains from the same sample (Empson, 1957, Table 2) it will be seen that in all cases Frit fly attack reduced the weight of kernel and, to a lesser extent, of husk.

TABLE 4

AVERAGE WEIGHT (MG), AND KERNEL CONTENT OF GRAIN DAMAGED BY FRIT FLY ATTACK

Class	Number of Grains in Sample	Husk	Kernel	Whole Grain	Percentage Kernel
Singles	19	7·4	7·4	14·8	50·0
Doubles					
Main	801	12·4	15·2	27·6	55·1
Bosom	141	6·4	11·5	17·9	64·2
Trebles					
Main	16	16·6	20·6	37·2	55·4
Second	7	9·3	12·9	22·2	58·1
Third	4	2·5	7·5	10·0	75·0

The loss in weight of fritted grains averaged about 50 per cent. There was an indication from other samples that this figure may apply only to slight attacks (about 5 per cent), and that, on average, damaged grains are lighter where the attack is more severe. This may be because, under heavy attack, many grains support more than one larva. As the attack falls most heavily on main grains the actual loss in a mixed sample of equal numbers of main and bosom grains is about 7 per cent for each 10 per cent of attack.

As Thomas (1948) has pointed out, the level of weight reduction is of little importance where fritted grains are all threshed out as tail corn. Thus, with oats for human consumption and for seed, every fritted grain is a complete loss. Because the attack falls most heavily on the main grains, the loss is normally about 12 per cent for each 10 per cent of attack but is rather less in proportion where the attack is very severe. For stockfood, however, preparation is less rigorous and feed samples have been seen with up to 35 per cent of the grains fritted.

There may be some loss of good grain during any additional cleaning required by heavily-fritted oats. The loss for each 10 per cent of attack may therefore vary from about 7 per cent where the attack is light and the oats are grown for stockfood, to over 12 per cent where an absolutely clean sample is required. In the first case, kernel content will be lowered, whereas in the second the differential removal of main grains may well cause it to be raised, the kernel content of bosom grains being higher than that of main grains (Empson, 1957).

THE EFFECT OF ATTACK ON THE CHEMICAL COMPOSITION OF THE GRAIN

Grain samples from the plots in four field trials were submitted for analysis to the nutrition chemists at the appropriate Regional Centres. In these trials different levels of Frit attack were obtained by spraying at various times with DDT miscible liquid. The trials were at : Kingsnorth, Kent, variety Star (3 per cent mean grain attack) ; Saredon, Staffordshire, variety Onward (5 per cent attack) ; Rosemaund, Herefordshire, variety Star (40 per cent attack) ; and Gleadthorpe, Nottinghamshire, variety Star (45 per cent attack). Samples of clean and fritted grain were analysed separately for the Saredon and Rosemaund trials, and from these results the composition of the difference between healthy and fritted grains (the theoretical lost material) was calculated. Only kernels were analysed for the Gleadthorpe trial and husk analyses from Rosemaund were used to complete the calculations. At this centre ether extract and soluble carbohydrate were not separated in the analyses.

At Kingsnorth whole samples with different levels of attack were analysed. From the results were derived regressions for the percentages of the various chemical constituents on degree of attack. Calculated values for healthy and attacked grain were then used to deduce the composition of the theoretical lost material.

The full results of the analyses of samples from Rosemaund are given in Table 5. The grain from Saredon (variety Onward) was a little higher in carbohydrate content and the composition of healthy kernels was, in this case, very similar to that given by Woodman (1957), whose results are summarized in Table 6.

TABLE 5

CHEMICAL COMPOSITION OF HEALTHY AND ATTACKED OATS, VARIETY STAR, 1953

	HEALTHY				FRITTED			
Percentage of Dry Matter of :	Main		Bosom		Main		Bosom	
	Husk	Kernel	Husk	Kernel	Husk	Kernel	Husk	Kernel
Crude protein ..	2·7	19·2	2·9	18·3	4·1	20·0	5·1	18·9
Crude fibre ..	46·1	4·1	42·5	4·2	48·1	25·1	47·2	16·0
Ether extract ..	0·2	5·2	0·2	5·2	0·3	3·5	0·3	4·9
Soluble carbohydrate	46·1	69·1	50·4	69·9	42·9	48·4	43·8	57·5
Ash	4·9	2·4	4·0	2·4	4·6	3·0	3·6	2·7

The slight superiority in crude protein content of the main grain over the bosom was also found by Berry (1920). It should also be noted that Hunter (1924) found real differences in kernel composition in variety Victory between crops grown on different soils in different years.

TABLE 6

COMPOSITION OF THEORETICAL MATERIAL LOST BY FRIT FLY ATTACK

Percentage of Dry Matter of :	Kingsnorth	Saredon	Rosemaund	Gleadthorpe	Oat Kernel (Woodman)
Crude protein	12·3	19·2	17·6	14·9	17·6
Crude fibre	3·2	1·3	1·3	2·4	2·1
Ether extract	7·4	6·4	5·3	80·5	7·0
Soluble carbohydrate ..	73·4	70·5	73·4		71·2
Ash	3·7	2·6	2·4	2·2	2·1

From the chemical analyses given in Table 6 it seems that the material lost through Frit attack is similar in composition to oat kernel, and this suggests that the main effect of attack is to prevent the full development of the kernels.

The accuracy obtainable by the analytical methods used is insufficient to indicate whether Frit larvae feed on the whole kernel, or whether they are selective in choosing mainly the carbohydrate fraction. The latter has been suggested by Gair (personal communication), and is supported by the high protein and low carbohydrate figures for fritted grain shown in Table 5.

CONCLUSIONS

It is now possible to suggest answers to the three questions posed in the introduction to the first of these two papers.

Does Frit fly attack on one grain lead to increased growth of adjacent unattacked grains ? There is no evidence of such compensatory growth and, in fact, there may be some reduction in both husk and kernel of adjacent unattacked grains. It may be noted that blindness, possibly caused by a lack or failure of fertilization, does lead to compensatory growth and increased kernel content of adjacent grains. The degree of blindness varies from crop to crop and with variety, but within each crop blind grains appear to be distributed practically at random and without relation to Frit fly attack.

Does the attack tend to fall on grains that differ in size from the average for the crop ? Main grains are damaged more often than the lighter bosom grains, especially at low levels of attack. Within the panicle, attack was found to fall most heavily towards the base. Grain weight decreased from apex to base so there is a tendency for the lighter grains to be attacked more often. The differences in attack and grain weight, however, are such that the error introduced is unlikely to be more than 1 per cent of the calculated loss. Attack was probably most severe on small panicles, but as there was no evidence of a constant relationship between grain weight and panicle size, no bias is likely to be introduced by variation in panicle size.

What is the difference in weight and chemical composition between attacked and healthy grains ? The mean weight of damaged grains from a lightly infested crop was about half that of healthy grains. With increasing attack the mean weight of damaged grains probably tends to be less, but a heavy attack falls proportionately more severely on bosom grains. The loss is about 7 per cent

for each 10 per cent of attack but may be at a rather higher rate where the attack is very severe.

Both husk and kernel of attacked grains contained, in proportion, slightly more protein and less carbohydrate than healthy grains. The analyses gave results consistent with the view that the main effect of the presence of Frit fly larvae is the prevention of the full development of the kernels.

The application of these results to the assessment of damage by the summer generation of Frit fly depends on the purpose for which the crop is grown. With crops for seed or for milling, each 10 per cent of attack causes a loss of about 12 per cent in weight, quality being unaffected or even enhanced. At high levels of attack the loss is proportionately less. There is, however, great variation in the normal loss during preparation of seed and milling oats (some seed samples were found to consist almost entirely of main grains only) and no fixed rate of loss from Frit fly attack can be applied to all such crops. For stockfood the loss in weight is about 7 per cent for each 10 per cent of attack. There is a corresponding loss of quality (feeding value) which is such that each 10 per cent of attack leads to a financial loss of about 10 per cent. It seems, therefore, that there is no appreciable correction necessary in the estimation of loss by the counting of attacked grains.

It should be remembered that this work takes no account of possible effects on grain and panicle size of attack to the shoots at the tillering stage, nor of damage to the panicles by larvae intermediate in time between the main first and second generations.

Thanks are due to Mr. W. I. St. G. Light, N.A.A.S., South Eastern Region (Wye), Mr. R. Gair, N.A.A.S., East Midland Region, the staffs of the Departments of Nutrition Chemistry, N.A.A.S., West Midland, East Midland and South Eastern (Wye) Regions and to the Directors of the Rosemaund and Gleadthorpe Experimental Husbandry Farms, for assistance in the section dealing with chemical composition.

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THE CONTROL OF *RHIZOCTONIA SOLANI* ON POTATO

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Routine disinfection of seed potatoes against *Rhizoctonia solani* Kühn, and other surface-borne diseases has been carried out in Scotland. Certain short dips, namely, for 15 minutes in 0·15 per cent phenyl mercury acetate solution in water, or in an aqueous solution containing 0·1 per cent each of phenol, mercuric chloride and common salt (with or without a wetting agent) and followed by a short rinse in plain water, have been used for some time. The efficacy of the treatments has, however, proved to be rather inconsistent as regards the control of *R. solani*. Therefore, studies were undertaken to determine the actual extent of control of tuber-borne infection of *R. solani* obtainable by these treatments as compared with the older dips in 0·1 per cent mercuric chloride, or 0·2 per cent mercuric chloride acidified with hydrochloric acid (Hough and Mason, 1951). Other dips were also tried in a search for quicker disinfection methods. Details of the treatments were as follows :

1. Phenyl mercury acetate (PMA). 15 minutes dip in 0·15 per cent solution of a five per cent commercial product in water.
2. Mercuric chloride. 90 minutes dip in 0·1 per cent solution in water, followed by a thorough rinse for 15 minutes in three changes of water.
3. Acidulated mercuric chloride. 5 minutes dip in 0·2 per cent solution of mercuric chloride in water plus 1·0 per cent hydrochloric acid, followed by a wash as in treatment 2.
4. Phenol-mercuric chloride. 15 minutes dip in an aqueous solution containing 0·1 per cent each of phenol, mercuric chloride and common salt, followed by a short rinse in plain water.
5. Phenol-mercuric chloride as in treatment 4, plus a wetting agent.
6. Ethoxyethylmercury chloride (EMC). 15 minutes dip in 0·5 per cent solution of a 6 per cent product in water.
7. Ethoxyethylmercury chloride plus a wetting agent. As in treatment 6.
8. Rimocidin sulphate (an antibiotic fungicide). 15 minutes dip in 0·01 per cent solution in water.
9. Hypochlorite. 30 minutes dip in 5 per cent solution of commercial product, acidified with $\frac{1}{10}$ of its volume of M sodium bisulphate, followed by a short rinse in water.
10. 2 : 4-dichloro-6-(O-chloranilino)-s-Triazine. 15 minutes dip in 0·1 per cent solution in water.

Tubers showing a number of sclerotia, which could be visually classified into negligible, thin, medium and thick types (Plate II, 2), were selected and dipped in the various solutions. This was intended to simulate commercial practice as against the studies of Guillemat and Lelièvre (1952) which were undertaken on detached sclerotia of one size only. The efficacy of the treatments was judged by observing the germination of the various categories of sclerotia on potato dextrose agar. Suitable controls were maintained. The results are shown in the following table.

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EFFECT OF VARIOUS TREATMENTS ON GERMINATION OF SCLEROTIA OF R. SOLANI

		Number of Sclerotia							
		Negligible		Thin		Medium		Thick	
		+	−*	+	−	+	−	+	−
1.	Phenyl mercury acetate ..	4	13	15	19	55	18	36	2
2.	Mercuric chloride	0	12	1	11	5	7	4	4
3.	Mercuric chloride (acidified) ..	0	12	1	10	7	4	6	1
4.	Phenol-mercuric chloride ..	0	7	6	6	10	2	10	0
5.	Phenol-mercuric chloride plus wetting agent	0	11	4	16	10	5	8	0
6.	Ethoxyethylmercury chloride ..	0	20	1	19	0	20	7	10
7.	Ethoxyethylmercury chloride plus wetting agent	0	20	0	20	1	23	2	13
8.	Rimocidin sulphate	8	1	10	0	9	0	8	0
9.	Hypochlorite (acidified with $NaHSO_4$)	2	10	14	5	14	3	24	0
10.	2 : 4-dichloro-6-(O-chloranilino)-s-Triazine	14	6	19	1	20	0	20	0
11.	Control (no treatment).. ..	17	7	35	0	26	0	28	0

* + = germination; − = no germination.

It was observed that all mercurial dips except PMA inhibited the germination of all negligible sclerotia; the thin ones reacted inconsistently to PMA and phenolmercuric chloride treatments but were virtually killed by mercuric chloride and EMC dips; the medium and thick sclerotia were, on the whole, relatively invulnerable. The inability of some of the untreated, negligible sclerotia to germinate in certain cases seems to indicate that some of them die out in the course of time owing to natural causes, such as desiccation.

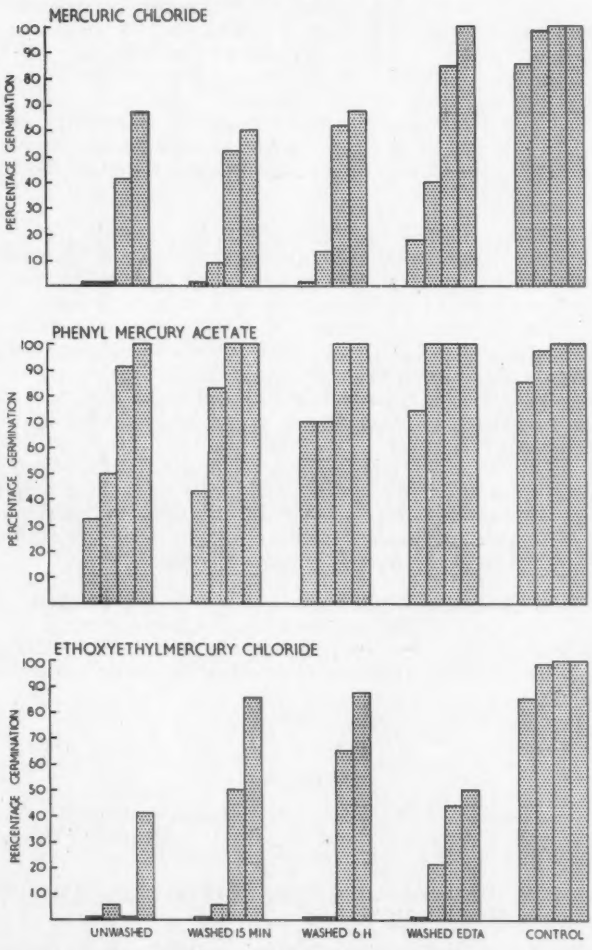
A preliminary experiment was also performed in which sets of tubers were left unwashed up to 16 days after treatment with PMA and the mercuric chloride dips, with a view to investigate the effect of any possible translocation and absorption of the chemical into the inner layers of especially thicker sclerotia in the course of time. No increase in the efficiency of the treatments was noticed.

It would appear, therefore, that until better products become available, the best chances of control of *R. solani* on seed potato tubers lie in their treatment with EMC solution plus spreader, or, failing this, with 1/1000 mercuric chloride solution for 1½ hours. If the tubers with medium and thick sclerotia are removed before dipping there will be even better chances of control. Treatments with acidulated mercuric chloride and PMA, too, would seem to be reasonably effective in such cases, but tend to be rather inconsistent owing to varying thickness of the sclerotia.

EFFECT OF WASHING AFTER TREATMENT

In the course of the work, it was noticed that if tubers treated with mercury compounds were washed in water longer than usual there was a decrease in the efficiency of the treatments. This appeared to indicate a fungistatic rather than a fungicidal action of mercury on the sclerotia. To investigate this point, separate sets of tubers after dipping treatments 1, 2, 3 and 6 were; (*a*) left unwashed, (*b*) washed in water for 15 minutes, (*c*) washed in water for 6 hours, and (*d*) dipped in a 0·1 per cent aqueous solution of the disodium salt of ethylenediamine tetraacetic acid (EDTA) for 5 minutes, followed by a short rinse in water. This latter treatment was included because EDTA is known to

chelate with mercury, and has been reported to revive *Alternaria* spores treated with copper fungicides (Müller and Beidermann, 1952). Between ten and twenty sclerotia of each category from each treatment (Plate II, 2) were plated out on potato dextrose agar to observe germination. The results are summarized graphically by the histogram in the figure below.



Effect of washing with water or EDTA on germination of sclerotia of *R. solani* on potato tubers treated with mercurial compounds. The four columns for each treatment represent, left to right, percentage germination of negligible, thin, medium and thick sclerotia.

It will be seen that washing in water did improve germination of even negligible and thin sclerotia treated with PMA, and of thin and medium sclerotia treated

with mercuric chloride. It also considerably increased germination of medium and thick sclerotia treated with EMC.

The effect of EDTA was more pronounced in some cases. An almost 100 per cent revival of negligible and thin sclerotia was noticed with PMA treatment. So far as mercuric chloride dips were concerned, EDTA partially reversed their effect on negligible and thin sclerotia, but increase in germination was very marked with medium and thick material. With EMC however, it seemed less efficient in bringing about a revival than mere washing in water. This may be because it is unable to chelate with the mercury atom in EMC. Also, EMC is likely to be removed more readily by washing in water for 15 minutes or longer owing to its high solubility. Considerable quantities of mercury were detected by us using a microchemical test (Cunningham and Anderson, 1954) on the sclerotia treated with EDTA, whereas very little was found on those washed in water.

Bodnar and Terenyi (1932) have pointed out that absorption of mercury into the protoplasm is necessary to bring about death, but that adsorption on the cell surface is merely inhibitory to growth. The observations reported here seem to indicate that the phenyl-mercury ions or acetate molecules are not absorbed by the cells of the sclerotium of *R. solani*. They are not even adsorbed on the surface efficiently, as mercury could be detected only on thick sclerotia, and its effect could be reversed easily by EDTA. On the other hand, with mercuric chloride or EMC, the mercury appears to be both adsorbed on the cell walls of the surface layers of the sclerotium, and absorbed into the protoplasm, the latter producing an irreversible action. This latter effect is very apparent with negligible and thin sclerotia, where all the cells are better exposed to the action of mercury. With medium and thick sclerotia, however, absorption occurs only in the outer layers, which are killed, but the interior cells retain viability. Germination of such sclerotia appears to be merely inhibited by the mercury adsorbed on the dead cells of the outer layers.

The practical significance of the above results is hard to estimate. However, it is possible that some of the apparently dead sclerotia when exposed to the various physical and chemical agencies in the soil, including leaching by rain water, might have sufficient mercury removed or inactivated to allow them to germinate.

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